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BACILLARY PHTHISIS

OF THE LUNGS

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BACILLARY PHTHISIS

OF THE LUNGS

BY

GERMAIN SÉE

PROFESSOR OF CLINICAL MEDICINE IN THE FACULTY OF PARIS

TRANSLATED AND EDITED FOR ENGLISH PRACTITIONERS

BY

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TRANSLATOR'S PREFACE.

Sir Thomas Watson, writing in the *Nineteenth Century* for May, 1877, says:—"The abolition of *zymotic* disease which our insular position would greatly facilitate is, then, a consummation devoutly to be wished, but it cannot be looked for in the lifetime of an old man in his eighty-sixth year; yet he may not be too sanguine in trusting that it will be witnessed in the next generation, or at least by his grandchildren." We who come after this Nestor of Medicine, minding well the difference that separates "a hewer of marble" from a Michael Angelo, do hope in some small way to help on a great work. We deal with the abolition or prevention of disease, when possible, often with a wider range than Watson as against the maladies then called zymotic.

Professor Germain Sée has done good service towards this end, and I feel happy to be able, by special permission, to place the results of his labours in the hands of English Practitioners.

London,
February, 1885.

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e. Common microbes coming from the mouth.

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a. Isolated tubercle-bacilli.

b. The same, forming a small group.

c. Mucus.

d. Epithelial cell from the mouth.

e. Cell from pulmonary epithelium.

f. Common microbes of the mouth : leptothrix and coccus.

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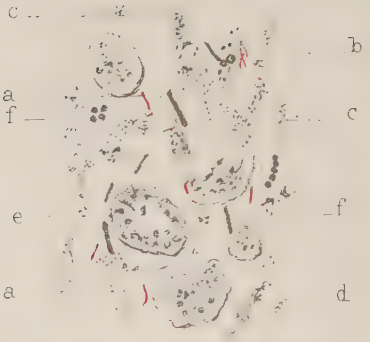
c. Arteriole, in the wall of which are certain bacilli.

d. Bronchus partly destroyed on the surface of the cavity.

Fig. 1.

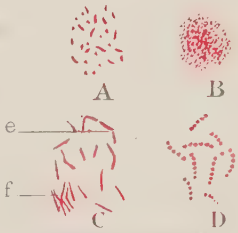
Phthisical expectoration with numerous bacilli

Fig. 2



Phthisical expectoration with few bacilli

Fig. 3

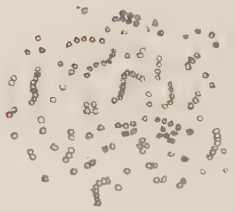


Tubercle bacilli

Fig. 4.

Microbes of vegetable infusions

Fig. 5



Coccus

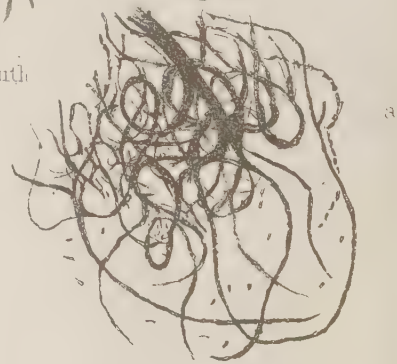
Microbes of the Mouth

Fig 6

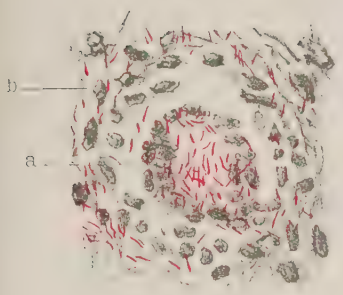
Fig. 7.

Bacillus anthracis. in the spleen.

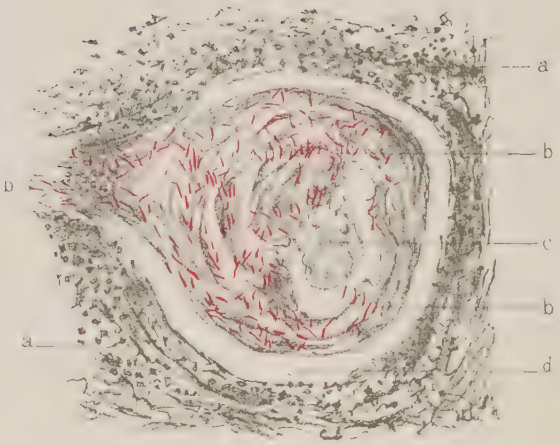
Fig. 8



Bacillus anthracis in cultivation

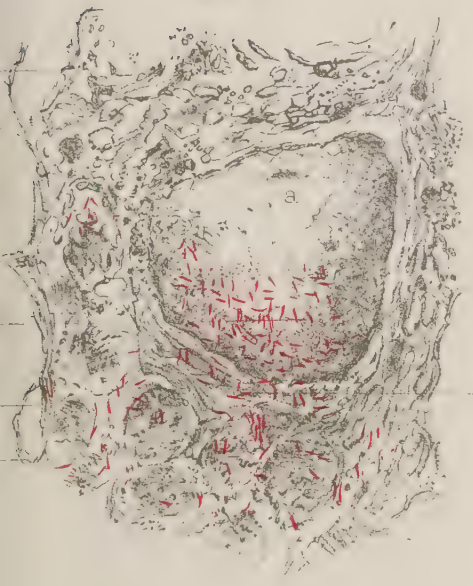


Giant cell from the tonsil



Fibrous tubercle

Fig. 1.



Miliary tubercle.

Pulmonary cavity

After Cornil.

Tubercle Bacilli in the tissues

BACILLARY PHTHISIS

OF THE LUNGS.

PRELIMINARIES.

I.

HISTORY AND TRADITION.

FOUR names have shed lustre on the history of phthisis, and they bring before us its summary to-day. *Laënnec*, whose immortal works have for sixty-five years established clearly its specific character, in the midst of a host of complex lesions which sometimes precede, often accompany, nearly always follow, and, so to speak, surround it, in such a fashion as to make the disease easy to be misunderstood. *Villemin*, who, by precise persevering experiments, demonstrated in 1865 the virulence of phthisis, and its transmissibility by inoculation. The third name, that of *Pasteur*, seems at first sight a stranger to the study of phthisis, but without his marvellous discoveries on the vitality of ferments, and especially of the parasitic origin of certain essentially virulent diseases, science would not to-day have to claim an exact knowledge of the mode of development of this, the most grave and most frequent of the maladies

which decimate the human species. *Koch,* in 1882, by new and rigorous technical methods discovered and described the parasite, the true agent of tuberculosis, in a manner which soon won general acceptance.

§ 1. *Bacillary Phthisis in its Relations with Medical Traditions.*

The science of diagnosis has already largely profited by the discovery of morbigenetic parasites, and we have contributed on our part the demonstration of doubtful forms of phthisis by the microscopic examination of the expectoration. When found in the *crachats* the bacillus is the constant and unexceptionable witness of tuberculosis in all its stages, even in those insidious beginnings which perplex the practitioner during long months of uncertainty; the bacillus, then, is an infallible sign, but that is not all. This parasite is further the proven promoter of phthisis, and the destructive agent of organs attacked by it.

If it is a bold innovation, it follows at least logically from the facts already acquired by science when we define phthisis by the bacillus which produces and characterizes it.

Morbid states of specific nature have long had a place in nosography. It has been assigned to them as their character, transmissibility, contagious nature, that is to say, an indiscernible, invisible principle. In place of a word, we have to-day real evidence. *Specificity,* long vaguely established, long under discussion, rests now on proofs that we can call *living.*

Our bodies are the theatre of the struggle between the life of man and that of the parasitic world, our hereditary enemy; the biological history of ferments and of disease-producing micro-organisms will constitute the modern doctrine of *experimental vitalism.*

II.

DEFINITIONS OF TUBERCULO-BACILLARY PHTHISIS.

Of the many ways of defining phthisis we shall take for guide and boundary its bacillary origin.

§ 2. *Anatomical Definitions.*

As Laënnec clearly established, tuberculosis assumes different forms which start all from the same sole lesion—the miliary granulation which is found in the lung of phthisical patients, yellow or gray tubercle which succeeds the granulation, the infiltrated gray or yellow mass which results from the fusion of tubercles, and, lastly, the caseous state and softening constituting the inevitable evolution—all these lesions, as well as the destruction of separate or confluent morbid products, are, with varied aspects and phases, of the same kind; it is the same tuberculosis.

Thus each of these forms has been successively discussed or denied. It will be necessary to appeal to the progress of histology to decide questions constantly recurring. But it must be remembered that one of our most eminent histologists, Professor Cornil, has publicly avowed the emptiness of established distinctions, and describes the complete analogy between tubercle, syphiloma, and the product of farcy. Such confusion cannot exist without damaging the scientific position of medicine. Tuberculosis, syphilis, and farcy constitute undeniable and perfectly defined individualities.

§ 3. *True Ætiological Definition of Bacillary Phthisis.*

But from the moment that anatomy is unable to decide the problem, we must seek another criterion. In tuberculosis the ætiological element is now rigorously defined;

it is the bacillary parasite which produces the tubercular lesion, and consequently also the functional trouble that ensues. It is against the bacillus that we should direct prophylactic measures for threatened individuals, and curative means for those attacked.

Phthisis is, then, a virulent malady, due to a special micro-organism, which is specific, always inoculable to animals, transmissible from suffering man to healthy men by way of direct contagion, but much more frequently by heredity, very frequently localized in a single organ, and thus to be cured without compromising the rest of the body. The bacillus, on the contrary, whilst it lives, or whilst it invades all the economy, whilst it multiplies there, constitutes the danger; it will continue its ravages.

§ 4. *Plan of Studies.*

1. Biological study of micro-organisms in general.
2. Biological study of the bacillus tuberculosis.
3. Anatomical study of bacillary lesions.
4. Study of causes.
5. Clinical studies.
6. Prophylaxis.
7. Therapeutic measures.

FIRST PART.

Biological Study of Micro-organisms in General.

III.

THE BIOLOGICAL STUDY OF THE MICRO-ORGANISMS OF TUBERCLE CANNOT BE UNDERTAKEN WITHOUT A PRELIMINARY NOTION OF THE PARASITIC KINGDOM.

§ 5. *General Characters, Functions, and Diffusion of Micro-organisms, their Vital Force.*

Their functions.—In the surrounding medium, at the extreme limit of visual apprehension, when reinforced by all the resources of optics, the observer finds organisms in quantities which cannot be measured. These, by their prodigious activity, their infinite multiplication, play an important part in the balance of nature and in the existence of man. They effect the destruction of inanimate organic substances by putrefaction; they provoke the most varied fermentations, and are thus, for the digestion of our food, the most indispensable auxiliaries. Other parasites invade animals of the lowest ranks as well as the superior animals and man himself. Living at the expense of their host, they take from him the nutritive elements and oxygen of the blood-globules, and produce thus degenerations, lesions, special maladies, and too often death. In every way they

play a destructive part, often for the advantage of nature, but nearly always to the detriment of individual humanity.

Their diffusion.—They are to be found everywhere in the exterior world, in the dust of the atmosphere, in the soil, in the water, especially in organic substances and in liquids exposed to the air. The same beings are found nearer to us in our habitations, in our food; they accompany us and seek us out. At all times and places they may be hurtful to us.

Their structure and nature.—All micro-organisms are formed by a single cell, comprising, most frequently, a cellular membrane, always protoplasm, never a nucleus. This plasma is colourless and without chlorophyll, which distinguishes them from the algæ (Van Tieghem). Their membrane is of a proteid nature (mycoproteïn of Nencki). In certain ferments it is formed of cellulose. It is this extreme simplicity of their structure which constitutes the difficulty of classing them in the different kingdoms of nature. The greater part, as *Cohn* has demonstrated, are of the vegetable kingdom. These are plants which have the most elementary structure, the most simple method of reproduction, but with a prodigious power of multiplication they are *microphytes* (schizomycetous fungi, Näg.). There are a few which are found to be allied to animal forms, and fewer still which remain without classification; but all are living, all affect the most varied forms and metamorphose themselves in the strangest manner.

Their generation.—Whatever be their form or dimensions, whether present with defined characters or in the state of germs or spores, it is proved to-day that they never arise in a spontaneous manner, and are never formed in a complete condition. The infinitely multiplied and constantly agreeing researches of *Pasteur* may be cited in favour of this opinion.

Their vitality proved by movement.—The quality that proves the vitality of micro-organisms is that they can execute spontaneous movements. One sees them start suddenly, turn round their extremities, move backwards or forwards. The movements have been attributed to special organs, cilia, but *Van Tieghem* considers the cilia as simple passive prolongations of the cellular membrane, the movements being due to the contraction of the protoplasmic bodies of the cell. According to *Engelman*, it is especially oxygen or light which produces or augments these movements, but when one constantly agitates the preparation they diminish or cease.

§ 6. *Morphology of Principal Microphytes.*

The species that interest us most are four in number.

1. *Micrococcus.*—The micrococci comprise simple rounded globular or ellipsoid cells, not united together, and whose dimensions do not often exceed five-tenths of a millimetre.

2. *Bacterium.*—The bacteria consist of long cylindrical cells which are united into small masses so as to form short and rather voluminous chains.

3. *Bacillus.*—The bacilli consist also of long cells united together, but the chains, instead of being short, are long and more slender than with the bacteria.

4. *Spirillum, leptothrix.*—This species consists of bodies in slender filaments, whether simple like the leptothrix or coiled spirally as spirillum.

Modifications of form.—According to Cohn, these forms are true *species* which always present the same characters and never change one into the other; an opinion which has been denied by *Naegeli*, and especially by *Büchner*. But what is certain is, that even in the state of complete development the bacteria and bacilli may offer certain modifications of form, ordinarily by degradation, but

without ever losing the physiological properties which characterize them. Thus bacilli break up into fragments under the influence of certain vital conditions. One sees the upper half of the bacillus in the normal state, while the lower half is broken up into very short particles, furnished with a resisting membrane; this peculiarity should not escape the observer who searches for the bacillus. The same process takes place when the bacillus finds itself in contact with certain irritating substances, or which are incompatible with the conditions of existence of the parasite; as, for example, *tincture of iodine:* when these cells find themselves afterwards in a normal nutritive liquid they take their specific form again rapidly, but when, on the contrary, unfavourable conditions persist, all the colony finishes by transforming itself into cells analogous to those of the micrococci, and which finally may lose all power of development.

§ 7. *Reproduction, Multiplication by Division or by Spores.*

Division. Fission.—All these parasites, especially bacteria and bacilli, multiply and are reproduced by two methods, generally by division, more rarely by the development of the particles called spores.

The division takes place in such fashion that the cell becomes constricted in the place of division, and this contraction rapidly augments at the same time that the cell increases in volume, until it separates in two parts, which rapidly attain the primitive dimensions of the parent cell. The number of these cells may increase to infinity, and certain parasites are developed by fission in a prodigious manner. Place some fresh meat in distilled water in contact with air; in a few hours the micrococci and bacteria which come from the atmosphere multiply so as to make the water muddy.

Modifiers of reproductive power.—The power of multiplication augments or diminishes under certain conditions, which are the same as those which preside over the nutrition of the parasites. Thus they multiply infinitely less under the influence of light, with the action of pure free oxygen, with very high temperatures, or when contained in certain chemical liquids, as disinfectants, which at a state of concentration annihilate the vitality of micro-organisms. Where chemical or physical actions cease, reproduction recommences, and is constantly accompanied by the consumption of organic matter as well as saline substances contained in nutritive liquids.

Spores.—The parasites possess also a much more powerful means of reproduction, that is by corpuscles produced in an endogenous manner in the cells. These are *durable* spores, which are observed especially in the regeneration of bacteria and bacilli. These spores, which are known by their power of refracting light and by a characteristic double contour, are round, or slightly oval; generally smaller than the cells that produce them, they are more able to resist the agents that are destructive to cells, more rebellious to the temperature, which may be raised to 110° C. or be lowered to —110° C. with impunity. They are also more refractory to boiling, which should be prolonged. At a given temperature in damp places or nutritive liquids, they are transformed into young cells; in freeing themselves from the cellular envelope they finish by taking the form of bacillary cells. It is in this form that bacilli are contained in the air. Their generative power is at present undoubted.

Degeneration and transformation into zooglœa.—It often happens that one finds in liquids used for the cultivation of micro-organisms a thick gelatinous layer, which has been developed by rest and crowding together of the cells or as a consequence of the transformation of

their membrane into a gelatinous substance. These gelatinous masses, containing whole colonies of micro-organisms, have received the name of zooglœa. They are always found in old culture liquids. It is not known whether they come from one or several forms of parasite, or whether the formation of this mucous protoplasm causes the death of its constituents.

§ 8. *Conditions as to the Existence of Microphytes.*

The conditions of the existence of microphytes claim an analysis as nearly as possible complete, because they open up new ideas to the practitioner on therapeutic indications, new horizons in the domain of hygiene or prophylaxis. It is an unedited chapter in preventive medicine. The point is to know what are the nutritive organic or mineral elements of micro-organisms, what are the gases, what the physical states of temperature and light, which favour or impede the life of these parasites.

Nutrition by organic and mineral compounds.—Differing from the algæ by the absence of chlorophyll, which serves directly in the constitution of cells, the microphytes draw their materials of formation and nutrition, whether proteids or hydro-carbons, from organic substances. The carbon compounds are, so to speak, all utilized, provided they are soluble in water.

Naegeli has prepared the following list of useful forms: sugars, glycerines, vegetable acids, benzoates, which are the true furnishers of carbon.

Nitrogen is taken principally from the albuminates on condition that they pass into the state of peptone under the influence of a special ferment; ammoniacal salts answer the purpose with difficulty, they may, however, with sugar, suffice for the life of ferments. Mineral salts are not less necessary for the nutrition of microphytes; sulphur is indis-

pensable, phosphorus not less so ; further, the alkaline bases, potash, lime, and magnesia, are useful. Thus the best culture liquids contain peptones and sugar with acid phosphate of potash, or they may be composed of sulphate of magnesia with leucin and sugar. Extract of meat contains in itself a certain number of potash salts. Different forms of nourishment exercise a certain influence on the vitality and properties of the organisms.

The bacillus anthracis may in certain culture-liquids lose its virulence (Büchner).

Life of micro-organisms with or without air.—Pasteur, Schützenberger, Traube, Brefeld, have begun by stating that microphytes do not undergo great increase but by assimilating much oxygen and eliminating carbonic acid; but Pasteur, after numerous researches, has come to the conclusion that if in the fermentation liquids there is any obstacle to the access of air, alcoholic fermentation is more active, whilst from the supply of oxygen it destroys but little sugar. In the other fermentations and in putrefaction, the same phenomenon is observed, and the process of fermentation has its maximum only in the complete absence of oxygen. Although the increase and multiplication of cells as well as their vegetative functions as ferments can be continued in atmospheres containing oxygen, yet there are functions and vital properties which cease. We have here the character of organisms which must be designated by the name of *anærobes*, in opposition to those much more rare forms which can neither exist nor perform their functions without atmospheric air, and which merit the name of *ærobes*.

However, this division is not free from all reproach, or rather it is not absolute; the fact is, the vegetative life and growth of the anærobes is not stopped by the oxygen of air, which with difficulty and by exception succeeds in

killing these parasites. It seems, then, an acquired fact in science, and a general law, that the most diverse vegetable cells have the power of forming products of fermentation (amongst others carbonic acid and alcohol) without atmospheric oxygen taking the least part in it.

Influence of temperature on the life and properties of micro-organisms.—The limits of temperature vary for *each* parasite, and also according to the quality of culture liquid and the presence or absence of oxygen. When the temperature is raised above a certain point cadaveric rigidity is produced. But even in this state life may re-appear under new conditions of nutrition; thus the bacillus anthracis recovers its infectious qualities in a slightly alkaline medium containing extract of meat after an hour and a half of rigidity.

In general microphytes die or cease to multiply between + 5° C. and — 18° C. some, as the bacillus tuberculosis, at + 30° C.

Cultivated at a temperature of 25° C. the bacillus anthracis preserves its virulent properties, but at 36° they diminish sensibly, and that at each generation. All microphytes may be easily killed by the necessary elevation of temperature.

*Action of temperature on spores.**—The formation of spores is singularly influenced by the temperature of the culture liquid, the bacillus anthracis cultivated at 35° forms its spores in twenty hours, at 18° nearly three days are required; and at 15° no more spores are produced (Koch). For reproduction from spores a temperature of 35° to 37° is necessary. But what the practitioner must remember is the enormous resistance of spores to the most extreme temperatures. Whilst bacteria and bacilli cannot in general vegetate below 30° or above 90°, their spores

* Temperatures are always given in degrees Centigrade.

resist 110° and often even a prolonged boiling for several hours. In the dry state they support a yet more elevated temperature. Exposed to the most intense cold, spores are often refractory. Miquel has seen them resist a temperature of 60° below zero, Pictet as low as — 110° C. It is this extreme resistance of bacillary and bacterian spores which constitutes the great difficulty in purifying air and freeing it from morbid germs. The glory of first taking practical measures against micro-organisms belongs to Lister, who in so doing revolutionized the surgical world.

IV.

BIOLOGY OF PATHOGENIC OR SPECIFIC MICROPHYTES.

§ 9. *Morbific Properties defined.*

In a normal state the bodies of men and animals contain different parasites; the mouth and the digestive tube always, but blood and urine only in certain morbid conditions, principally with infectious maladies. These specific diseases are absolutely distinct from mineral or vegetable poisoning, which, whether in the acute or chronic state, have always effects proportional to the dose of the poison. For the malady, the type depends on the species of microphyte from which it is derived.

§ 10. *Conditions of Existence of Morbific Microphytes.*

Difficulties of existence.—In general the conditions of the existence of specific microphytes are not favourable for their life and multiplication; that is why they are relatively rare if one compares them with the parasites generally so widely diffused. If pathogenic micro-organisms had multiplied like those producing fermentations, the population of

the world would long ago have perished from infectious maladies. The universe would have been for the micro-organisms, they would have devoured all organized bodies up to the extreme limit, and this pasture once used, the world would have been deprived of animals and plants. Happily these pathogenic forms suffer in the struggle for existence, and must find a suitable soil to produce a contagious malady.

Influence of oxygen.—All morbid microphytes can live and do live without air; they multiply when one suppresses or diminishes the access of oxygen. They cannot bear free air, and under the influence of the atmosphere they even lose their morbific character.

Influence of heat and motion.—A certain temperature is necessary for specific microphytes to multiply; below or above this determined limit they lose their power of reproduction. They may either die or develop into spores. Violent movements of the culture liquids arrest the vitality of the microphytes. It is easy to understand the conditions of germination of micro-organisms; carcases putrefying in the earth, or on its surface, favour the development of ordinary microphytes; specific parasites are only developed when the dead bodies contain their species; in this case the multiplication of the morbid parasite may be such that the fermentation organisms find no more source of nutrition.

The soil itself does not favour the formation of micro-organisms, but when the cadaveric matters are of a gelatinous nature. *Water* may be altered by passing through a soil saturated with animal matters. We know, too, from Schlösing that oxidations take place in the soil from animated ferments; for example, the ammonia which results from certain decompositions is transformed into nitrous acid, and the nitrification of the soil is, so to speak, animated. Boussingault has shown that a soil saturated

with humidity and containing but little air is not susceptible of nitrification. *Habitations* constitute in themselves a manifest source of micro-organisms of all species. They always find sufficient nutrition in the *debris* of the sub-soil, and in the rooms where they penetrate with organic dust they stick to the furniture and the paper hangings, the latter ordinarily saturated with the nutrient material used to make them adhere to the walls.

§ 11. *Morphology of Specific Microphytes.*

Specific microphytes differ essentially from those producing fermentations by their pathogenic properties ; the cultivation contrived by Pasteur leaves no doubt on this point. It is not the same as to the forms in which one or the other appears. Morbific agents as well as ordinary species take the shape of micrococci, bacteria, bacilli, spirillæ. If bacteria and bacilli predominate in pathogenesis, it is not less true that they are sometimes not of a disease-producing order. Micrococci are found in the ordinary state more than the morbific. But at least in the maladies which result from them can we find definite distinctive characters which enable us to recognize the two classes of microphytes by microscopic inspection ? This question of the specialty of forms can be answered in the affirmative in the greatest number of cases. By means of the colouring processes imagined by Weigert and perfected by Klebs, Koch, and others, we can assign, especially to bacilli, absolute characteristics. The forms of micrococci, their grouping and envelope, often enable us to recognize their intimate nature ; thus micrococci are easily coloured by solution of hæmatoxylin, whilst bacteria are with difficulty impregnated with this colouring matter. Certain bacilli retain aniline colours very powerfully, others easily abandon these colours, thus they are decolourized by acids or by a secondary treatment change

the first colour for the second. These peculiarities cannot but come from the permeability of the envelope for certain colouring matters. This may be considered the best answer to objections which have been formulated against the doctrine of virulent germs, which, according to certain physiologists, are but ordinary bacteria developed in an affected organism.

Spores.—There are always to be found spores coming from different species which are identical in form, or at least difficult to distinguish. Morbific spores differ but little in form from ordinary spores, and as to their specific properties they are difficult to demonstrate.

V.

MICROPHYTIC MALADIES.

Comparative medicine.—This was the first to profit by the discoveries of Pasteur. The parasitic nature of anthrax, cholera of fowls, typhoid fever of pigs, rot in sheep, have been clearly recognized.

Surgical and puerperal affections.—Amongst the maladies which have been the object of investigation there are those which have been demonstrated as microphytic by practical methods in the most peremptory manner. Such is surgical septicæmia, which is cured or prevented by antiseptics, though for its cause we seek definite microphytes. Such is puerperal fever, which, according to Doléris, is caused by several micro-organisms and is treated efficaciously by such antiparasitic remedies as iodine or corrosive sublimate. The parasitic theory is still in dispute, but not the curing of patients. This has been superabundantly proved by the magnificent results which Tarnier has obtained in that Maternity (Paris) which has been so much execrated because it was exposed to all these dangers.

Medical cases.—At present medicine has no reason to be proud of its antiparasitic therapeutics. We must take into account *all* the specific characters of parasitic maladies.

§ 12. *Classification of Parasitic Maladies.*

We shall take into consideration the origin of the malady, its mode of development, and its powers of transmission. With the assistance of these principles we shall harmonize the new discoveries of science with medical traditions. Five classes of parasitic maladies will be distinguished.

First Class.—*Miasma, malaria.*—We commence at first with malaria, which is simply of miasmatic origin, and not transmissible from man to man. Malaria and the palustral fevers constitute by themselves alone the first group, perfectly distinct from all other specific maladies. They are developed under the influence of miasma evolved from a marshy soil, or from earth rich in organic materials, as the Roman Campagna. Now in these cases the air and the soil contain bacilli, which, in culture liquids, take the form of recurved, chambered, or partitioned threads. Klebs and Tommasi Crudeli assert that these microphytes can be inoculated to rabbits, which is very doubtful. Malaria is neither transmissible, contagious, nor inoculable; it is miasmatic.

Second Class.—*Microphytic maladies which are inoculable, and at the same time transmissible by the air.* —There are five parasitic maladies.

1. *Tuberculosis.*—Amongst inoculable maladies it may seem strange to count in the first rank tuberculosis, of which but lately the virulence and contagion were contested. We know now that it is due to a special microphyte, to a bacillus easy to distinguish from inoffensive neighbours, not only by its power of indefinite multiplica-

tion, by its inoculability, but also by technical characters. The vital properties discovered by Koch neither permit nor encourage confusion. It is to be found in all the tissues, more frequently still in the expectorated matters, and probably in the air which has been in contact with them.

2. *Variola.*—Small-pox is equally transmissible by air and by inoculation of its quadrigeminal micrococci (Klebs), which accumulate especially in the little areolar cavities of the Malpighian layer at the place of the pustules (Cornil and Babès).

The solid particles of vaccine have been well shown by Chauveau to be the active agents. They are now known to be microphytes.

3. *Diphtheria.*—According to Talamon, this fatal malady presents a special mycelium, under the form of divided refractile tubes and conidian spores, which may be cultivated and injected with success to pigeons and young cats.

4. *Recurrent fever.*—This malady, which is unknown in France, is due to a perfectly defined spirillum, which is found in the blood only at the times of attack. Its inoculability is doubtful (Obermeier).

5. *Erysipelas.*—In its infectious forms isolated or conjoined micrococci are to be found, particularly in the lymphatic trunks, and in the lymphatic spaces of the derma. They do not exist in the blood, and their reproduction by inoculation has not been obtained by Felheisen.

6. *Contagious pneumonia.*—The infectious character of most inflammations of the lungs can be no longer doubted (see our researches, *Union Médicale*, 1883). According to Friedländer, the infectious nature is caused by micrococci, furnished with capsules; and, after Talamon, by ellipsoid micrococci, which, by themselves alone, determine fibrinous pneumonia.

7. *Typhoid fever.*—Rarely transmitted by contagion

from one individual to another, Jaccoud has shown it to be carried, twice out of thrice, by the agency of fæcal matters, especially when they are mixed with typhoid stools. Water and milk serve also as a vehicle for the typhoid virus. It consists of bacilli, which Eberth has found eighteen times out of forty in the intestinal glands; further, in the larynx and lungs, in form of bacteria. Bouchard has found it in the urine, and Hanot in the roseolous eruption.

THIRD CLASS.—*Microphytic maladies which are only inoculable.*

8. *Rabies.*—Which has been recently investigated by Pasteur.

9. *Syphilis.*—Aufrecht says this malady, so easily inoculable to man, is characterized by diplococci which are strongly coloured by fuchsine. Klebs has cultivated them, but there has been little success in reproducing the malady amongst animals.

10. *Gonorrhœa.*—Neisser has described a special micrococcus which is to be found in the urethral discharge and the pus from gonorrhœal ophthalmia. The product of culture has been inoculated with success.

11. *Glanders. Farcy.*—This is a very grave virulent malady. The virulent character is due to a bacillus very like that of tuberculosis, but which is to be distinguished by colour reactions (Bouchard, Capitan, and Charrin).

FOURTH CLASS.—*Non-inoculable parasitic maladies.*

12. *Lepra.*—A bacillus like that of tuberculosis is to be found, but it is not inoculable.

13. *Ulcerating Endocarditis.*—Here the bacteria form veritable colonies in the blood, in the heart, and in the urine. They have not yet been cultivated.

14. *Rheumatism.*—According to Klebs, this malady is also of parasitic nature.

Fifth Class. *Contagious maladies whose parasite is doubtful or badly defined.*

15. *Measles, Scarlet Fever.*—Micro-organisms still unknown.

16. *Whooping Cough.*—Whooping cough, which by its contagion and its localization in the lungs has so much affinity with measles, is characterized by a mycelium and spores, which after Tschauer present a special type. They have not yet been cultivated.

17. *Dysentery.*—The micrococci and bacteria which Radjewski has found in the fæcal matters are nothing special; the same microphytes are found in the normal state.

18. *Cholera.*—The French and German Commissions have not yet obtained exact knowledge of the parasite of cholera, of which the contagion is but little in doubt. However, Koch recognized as cholera-producing certain recurved bacteria, though he has not been able to cultivate them.

TRANSLATOR'S NOTES.

Professor Lionel Beale says in his work, "The Microscope in Medicine," fourth edition, p. 145, "Futile will be the determined and repeated efforts to force people to believe that these ever-present, growing, and multiplying bacteria are actually *disease germs*. The evidence is unsatisfactory and many of the statements untrustworthy." This opinion, I believe, is still largely shared by many English practitioners, and in Paris two of the ablest professors (Jaccoud and Peter) may be cited as teaching that bacteria are only carriers of contagion. Hallopeau ("Pathologie Générale," Paris, 1884), after noticing the remarkable article, "Germes," by Ch. Robin, in the *Dictionnaire Encyclopédique des*

sciences médicales, says "It is impossible to mistake the significance of reservation in opinions emanating from such eminent pathologists; they do service in forcing the partisans of the new theory not to venture into the path of hypothesis, and only to admit as demonstrated facts supported by absolutely certain proofs." Bouchardat considers that the parasite of tuberculosis does not exist in nature outside the human body, but this can scarcely apply to the spores.

The reader would do well to give some consideration to the communications of Pasteur and Koch in the Copenhagen International Medical Congress.

Valuable information is also to be found in an original communication by Mr. Watson Cheyne, *Practitioner*, April, 1883, being a "Report to the Association for the Advancement of Medicine by research on the relation of Micro-organisms to Tuberculosis."

SECOND PART.

Study of the Bacillus.

VI.

FORMS AND BIOLOGY.

§ 13.—*Morphology of the Bacillus Tuberculosis.*

Amongst microphytes the bacillus tuberculosis interests us most, because, although scarcely discovered two years, it is the best demonstrated, the most clearly characterized.

I. *History of tubercular microphytes.* — Some years before the discovery of the bacillus, Klebs made important researches as to the cultivation and inoculation of the parasitic agent. These were verified by different observers in France, Germany, and in England.

Zooglœa.—In 1881 Rindfleisch and also Aufrecht believed they had found in the centre of the tubercle, in what are called giant cells, those microphytic masses which we have described under the name of zooglœa. Malassez and Vignal cultivated and inoculated these zooglœa. They found that at the third generation of tubercles, which result from the injection of these zooglœic liquids, the form of *bacilli* was resumed. It seems, then, they are only a transitory form of the bacillus.

Bacilli.—These were indicated by Baumgarten, though he did not know how to show them. It was Koch who, by

a special colouring process, succeeded in demonstrating the microphytes. In the month of April, 1882, he communicated to the Medical Society of Berlin the results of his researches, and proved in a conclusive manner that in the tuberculous organs of man there exist corpuscles arranged in chains, which by their chemical and morphological properties differ totally from all other known forms of microphyte. They are the bacilli of tuberculosis.

II. *Descriptive characters.* — In very slender chains, whose length equals hardly one-fourth or half that of a red blood globule; they resemble, principally in form, the thinner and more pointed bacilli of lepra. Amongst the bacilli spores are often found, that is to say, fine, rounded, refractive bodies. Also side by side with the bacilli are found the gelatinous masses known under the name of zooglœa (Rindfleisch, Malassez, Renaut).

§ 14.—*Technical Details.*

1. *Koch's method.*—The bacillus can only be discovered by the process of colouring invented by Weigert and perfected by Koch, who demonstrated that the bacillus once coloured by methyl blue could not be coloured by vesuvine, whilst the other microphytes in the preparation lost their blue colour to take the brown tint of the second colouring matter.

2. *Ehrlich's method.*—Ehrlich remarked that the bacillus impregnated with an aniline colour resisted the action of dilute nitric acid, whilst the other elements, micro-organisms and cells, are immediately decolourized. He employs the solution of Weigert, thus formulated:

Saturated watery solution of aniline, 100 c.c.
Saturated alcoholic solution of fuchsine, 11 c.c.

The following description is by Talamon: "The tissue or expectoration which one wishes to examine is spread out in

a thin layer on a slide; it is allowed to dry in the air. Afterwards the slide is passed two or three times into the flame of a spirit lamp to coagulate the albumen. The slide thus prepared is placed for from twelve to twenty-four hours in a glass vessel containing the colouring fluid. It is then washed with distilled water, afterwards in a solution of nitric acid (30 per cent.), until all the colouration has disappeared. It is washed again with distilled water. The slide is then immersed in a watery solution of methyl blue. The preparation is dried and mounted in Canada balsam. It shows the bacilli strongly coloured red, the cells with their nuclei and the other micro-organisms being coloured blue." This proceeding, modified by Rindfleisch, who warms the colouring solution slightly to make the staining of the bacilli more active, has been approved by Koch himself. Numerous modifications have been introduced into these technical methods, such as those of Gibbes or Fräntzel, but the most universal and surest method is that of Ehrlich.

§ 15. *Biology of the Bacillus. Its parasitic life.*

The bacillus is found not only in true phthisis, but in people called *scrofulous*, who are attacked with osseous, glandular, and cutaneous maladies. The bacilli are to be found equally in a great number of tumours, up to the present undetermined in character, which occupy the extremities, or even the viscera themselves, and in individuals in appearance perfectly healthy and exempt from all scrofulous or tubercular taint. There are also the same bacilli in the tubercular malady which affects the bovine race. We shall speak of it again with regard to danger from the flesh and milk of animals.

I. *Tuberculosis. Diverse lesions.*—Bacilli are to be found in the different forms of tubercular lesions, whether localized in the lungs or disseminated at the same time in

the other organs. When localized in the lung, the bacillus characterizes equally the first periods of the malady as well as the most advanced. It is to be found in the lesions called granulations and miliary tubercles, as well as in the destructive phases of caseation or ulceration. In all these cases its favourite, but not exclusive, habitat is in the tubercular elements called epithelioid cells and giant cells. The facts as to the presence of the bacillus have been ascertained by necropsy.

Liquid secretions.—During the life of the patient we recognize the microphyte with the same facility, the same constancy, in the products of expectoration. In masked, latent forms of phthisis the presence of the bacillus in the expectoration is a certain means of diagnosis.

Urine. Alvine matters. Blood.—The expectoration is the only secretion which constantly contains the bacillus. Urine and fæcal matters contain it in an exceptional manner when there is a tubercular lesion of genital organs or intestines. As to blood, it is neither a favourable nor habitual receptacle for the bacillus.

II. *Local tuberculosis, generalized later.*—Suppose pulmonary tuberculosis, at first clearly limited; from this central source the bacilli may in multiplying penetrate into the blood, or lymphatic system, and produce general tuberculosis (miliary). In these miliary tubercles, which are often found in the meninges, the pleura, the peritoneum, the liver, the kidneys, etc., the bacillus is to be seen, but in much less quantity than in the more or less advanced products of tuberculosis of the lung.

III. In those local tuberculoses which do not become general, as in the genital organs, the bacillus is evident although rare; and in this class spontaneous definite cures are often found.

IV. *Scrofula in general.*—In the bodies of those patients

whom we call scrofulous, without having defined their malady, constitution, or its predisposing causes, a bacillus is often found which by its identity with the bacillus tuberculosis may be regarded as proving also the identity of scrofula and tuberculosis.

Scrofulous Adenitis.—Also in scrofulous glands one frequently finds the caseous degeneration that we find in tubercular pneumonia. This caseous mass always contains the bacillus, so we can consider the disease external, localized tuberculosis.

Scrofulous Ostitis.—In bones attacked with ostitis, periostitis, white tumours, chronic fungous arthritis, the dominant lesion is tubercle, for the bacillus never fails.

Lupus.—Cornil has found bacilli, in small number it is true, but constantly in lupus which should henceforth be placed in the list of scrofulo-tuberculous maladies, and indeed Besnier has shown with sagacity at the Hôpital St. Louis that lupus endangers life by tubercles which it develops in internal organs, notably in the lungs.

V. *Local tuberculosis of different organs.*—It is not necessary that these lesions should be grafted on the so-called scrofulous soil. We see circumscribed, curable tuberculosis of the male and female genital organs. Affections of superficial mucous membranes, as of the pharynx, mouth, larynx, appear as lesions, the nature of which is unknown, at least, unless the examination of the products by the microscope is practised; the presence of bacilli shows categorically their tubercular nature.

VI. *Local tuberculosis becoming infectious.*—We must not forget that these circumscribed centres in bone, genital organs, etc., may, like those of the lungs, become the starting-point of a true infection which attacks the important organs of life. After a long series of dormant years these tubercular centres may, without known cause, pour into the

blood, bacillary colonies which ravage the whole organism. It is the familiar history of acute phthisis which has for its starting-point a bacillary source, so to speak, *forgotten*.

§ 16A. *On the culture of Microphytes, especially Tubercular Bacilli.*

Culture liquids.—Specific microphytes are developed with difficulty in the air, more easily in inert substances, ordinarily in living organisms. If they come from without it is by different ways of absorption, especially by respiration and alimentation, that they penetrate into the economy; there they form a circumscribed centre of development, or, multiplying, they are carried by the blood torrent into different parts of the organism. Then from these primitive or multiple centres or from the blood itself microphytes may be collected, put into a nutritive fluid, and there cultivated without losing their deleterious properties. It is only by indefinite culture in nutritive liquids constantly renewed, or, better still, by access of free air, that their pathogenic power is diminished.

Favourable composition of culture liquids.—The most nutritive substances for pathogenic parasites are not albuminous substances, but gelatine compounds, glutin, chondrine, and without doubt also mucine. We may use with advantage for culture, solutions of gelatine and decoctions of meat containing gelatine. In other culture liquids destined for putrid parasites, pathogenic bacteria lose their property and form. These gelatinous media are also to be found in nature, especially in the soil and stagnant water. But carcases and organic matters which putrefy on the surface of the earth constitute a bad centre of development for specific micro-organisms, because they soon lose their pathogenic properties by access of air, and in the second place they are soon replaced by ferment-producing forms.

Culture at a certain temperature.—For specific forms the temperature must be from 25° C. to 41° C., whilst the organisms of putrefaction develop between 16° and 20° C. and multiply at 15° C.

§ 16B. *Culture of the Bacillus Tuberculosis.*

Primitively Koch tried gelatine (extract of meat, peptone, and gelatine) without success, because the bacillus cannot grow at 20° C. the temperature at which gelatine melts.

He thought then of coagulated and sterilized serum of blood, which will furnish transparent layers.

The blood of the ox or sheep is warmed for six days, every day an hour at 58° C., afterwards several hours at 65°, so that the serum should remain coagulated. On this field of cultivation we sow particles containing bacilli, and expose it afterwards in an incubator to a constant temperature of 37° C. Towards the tenth day there are formed on the surface of the serum little scales and points. After several weeks, the growth of the bacillary colony is finished. Bacilli thus cultivated have often passed through eight or ten such operations before serving for experiences of infection, inoculation, or inhalation.

THIRD PART.

Anatomical Study.

VII.

ON TUBERCLE.

To justify our definition we have to demonstrate the facts dominating and resuming the whole question. (1) That it is the bacillus that produces the entire series of lesions that may be referred to tuberculosis. (2) That tubercle, considered solely from an anatomical point of view, without taking count of the bacillus, is a lesion susceptible of different explanations, and presenting no true characteristics. But before the proof we must give the descriptive anatomy of tubercle constituting a preface to the discussion.

§ 17. *Anatomical forms of Tubercle.*

Tubercle is formed by a clearly defined projection, visible to the naked eye, and called miliary granulation or tubercle. This body is itself composed of little microscopic centres formed each by a compact clearly limited mass of cells. These, however, when mixed with inflammatory products are often confounded in a general affection called tubercular infiltration.

I. *Histological character of primitive tubercle.*—Isolated or lost in the common affection, the microscopic nodule has

been considered as a neoplasm composed of cells, and nuclei of new formation, deprived of blood vessels (this is an important sign), and containing the characteristic giant cells which are protoplasmic masses containing twenty to a hundred nuclei. But we soon perceive that the structure is far from being so simple, so clearly defined, and that none of the elements are characteristic.

(a) *Epithelioid cells.*—Miliary tubercle in the primitive state consists of a mass of embryonic cells; some resemble *endothelia,* and possess great oval nuclei with brilliant nucleoli. The greatest number, however, have the same form as epithelial cells, and especially at the periphery of the nodule a certain number of lymphoid cells, like the white blood corpuscles, are to be found.

(b) *Giant cells.*—These were discovered by Wagner and studied with great care by Schüppel. These giant cells form an important element in the structure of tubercle; there are always two or three to be found in each mass, generally round or with protoplasmic prolongations. But these giant cells are to be found in other pathological products and even in normal tissues. Robin has found them during the physiological absorption of bone, Friedländer in the uterine blood cavities at the spot of the placental insertion. Heidenhain has seen them in the peritoneum after the introduction of foreign bodies, Friedländer in pneumonia, Heubner in syphilitic endarteritis, Baumgarten in gummata, Buhl and Jacobson in granulation tissue of wounds, Johnes and Pflug in sarcomatous tumours and in the foci of actinomyces. In spite of the multiplicity and ordinary character of the conditions which favour their development, these giant cells constitute an important element in the tuberculous nodule.

(c) *Absence of vascularity.*—There are only a few capillaries which penetrate from the compressed vessels of the

periphery. It is to be remarked that when these peritubercular vessels are obliterated by endarteritis the centre of the tubercle degenerates and becomes caseous.

(d) *Bacilli.*—These organisms are to be found generally in the giant cells, and constitute the dominant character of tuberculosis.

II. *Caseous tubercle.*—Tubercle, from its fatal tendency to caseous degeneration, has been defined as "a cellular projection deprived of vessels, and which at a given moment of its development becomes caseous." Although this change is not constant in man and rare in animals, Laënnec writes, "one sees a small, yellowish white, opaque point develop in the centre of each tubercle, and, growing from the centre to the circumference, invade the totality as it grows." Thus tuberculosis constitutes, according to Laënnec, under the various forms of granulation, isolated tubercle, and tubercles agglomerated and infiltrated into the tissues, one single identical affection which terminates infallibly by caseous change, that is, by fatty degeneration and process of ulceration. The fatal cause of this change is in the state of vital misery, the natural weakness of tubercular cells, especially where more or less insufficiently nourished by irrigation of blood. This fatty degeneration in great collections of tubercles, as in the parenchyma of the lung, gives rise by softening to the formation of cavities, and on mucous surfaces to an ulcerated state.

Fibrous tubercle.—Grancher and Renault show that whatever be its form tubercle always contains a fibrous zone in miniature, which, if the development of the tubercle is slow or arrested, may increase and lead to a natural cure.

§ 18A. *Evolution of Tubercle.*

1. *Histogenesis.*—Virchow thought that the granulations

were derived from connective tissue, but he was wrong. Cornil and Ranvier as well as Rindfleisch recognize that they may originate from epithelial cells—as, for example, the pulmonary alveoli. Colberg and Aufrecht assign to the giant cells and to the epithelioid cells an origin from the adventitious tunic of the vessels. What is certain is, that when the granulation surrounds a vessel, the inner coat of the artery inflames and produces giant cells. Ziegler has made it probable from his researches that the granulation may be developed from leucocytes escaped from the vessels. Once formed, the granulation presents a central zone of granular matter and a peripheric zone formed of compact embryonic cells which spread far enough into the surrounding tissues; in the centre, ordinarily destitute of vessels, are to be found the giant cells. By the side of those adult granulations, according to Grancher, appear young microscopic nodules and an irregular mass of cellulo-embryonic tissue having the same structure and destiny as tubercle. The growth of tubercle by proliferation of its elements is very limited, the augmentation of granulations being caused by new ones coming from the periphery.

II. *Local tuberculosis.*—Contrary to general experience of tumours, the infection of regions is not due to tubercular cells, but it is produced by the bacillus.

III. *Means of transmission.*—The lymphatic vessels are the principal means of transport for the infection, and a secondary tuberculosis of lymphatic glands results. Later on it may gain neighbouring organs—as, for example, a serous membrane. By means of the thoracic duct the blood circulation may be invaded, but this also takes place by effraction. Weigert has traced the tuberculosis of pulmonary veins and shown that a great many granulations are developed in this way. The granulations may be primitive or grafted on to a chronic tuberculosis remaining local and

latent. In general a caseous centre is found in a lymphatic gland or the source is in the apex of the lung from which the general tuberculosis starts (Bühl).

IV. *General tuberculosis.*—It is to be remarked that the primitive effect of the bacillus is purely local. It provokes special changes, and it is not until these derange the functions of the organ that the rest of the body is affected. It is quite clear that if tuberculosis is localized in the lung or in the brain it compromises life by the anatomical and functional relations of these organs with the whole economy. If the organ attacked is less important it may become the starting-point of a true general infection by the means we have traced.

§ 18B. *Evolution of Pulmonary Consumption.*

Pulmonary tuberculosis commences generally by the bronchi, the peri-bronchial tissue, and continues by the lung. But often a few microphytes in the lung itself produce inflammation, which spreads quickly and rapidly assumes a caseous character. Clinically, we may recognize different conditions, as the primitive granulations, caseation, necrotic, cavernous, or ulcerous *destruction;* lastly, fibrous transformation (sclerosis).

I. *Bronchitis.*—Tuberculosis commences usually in the walls of one or more of the smaller bronchi, and especially those that occupy the apex of the lung. It is on account of their minimum participation in the respiratory phase that the bacilli are more easily stopped at the apices of the lungs.

II. *Peri-bronchitis.*—From the bronchial surface the tubercular infection gains the periphery, and from this primitive source, where slight ulceration is soon established, the infecting substance easily reaches other bronchi through the medium of the air. In this peri-bronchial centre, often

to be recognized with the naked eye, we recognize the air passage in the midst of grayish, afterwards yellowish, masses. The air passage is ordinarily soon completely obliterated by the tubercular mass, and the termination dilates under the form of an irregular cavity, the first degree of *cavernous formations.*

III. *Lobular pneumonia. Atelectasis.*—The alveolar tissue is attacked in its turn, and is modified in different fashions. Pulmonary atelectasis is caused by the permanent obliteration of a bronchus, which produces the collapse of the corresponding lobule.

Caseiform lobular pneumonia.—This often attacks the weakened lobule, the alveoli being filled with pus corpuscles and large epithelioid cells, which, without doubt, come from alveolar epithelium. Finally, the caseous tissue falls into a state of necrosis, and forms in its turn new cavities.

IV. *Caseous pneumonia. Yellow and gray infiltration.*—On the other hand, these different lobular centres end by being united, the infiltration spreads more and more, so as to form gray infiltration (Laënnec), vitreous (Grancher), afterwards yellow infiltration, which indicates the caseation of this tubercular mass. It may be noted that caseous pneumonia is inflammation grafted on to the granulations.

V. *Caseous, calcareous degenerations; softening.*—These caseous collections, which are easy to distinguish by their yellow colour, their granular and fragile consistence, come either from lobal pneumonia, or from lobular inflammations fused into a single mass. They are in reality products which have become fatty and dry, also perhaps mixed with calcareous salts, capable of transforming the collection into a stony mass. It is a sort of cure for starting from this calcification, they are only inert bodies in the lungs. It

may also happen that around this pulmonary concretion are formed centres of softening, which allow the stone to be detached and to be expelled.

VI. *Formation of cavities.*—These are formed when the liquefied caseous mass is transformed into pus and expelled through one or more of the bronchi, leaving in its place an empty space, which is afterwards filled again. The inner wall is at first irregular, corrugated, and jagged, but often several cavities unite. They have always a tendency to gain the periphery, although interstitial sclerosis commences, and, at last, surrounds the cavity with a capsule of connective tissue. When the contents of this pouch are destroyed by suppuration and expectorated, the primitively villous wall of the cavity is transformed into a smooth surface, which presents here and there a caseous mass, granular and easy to be detected. Cavities are also formed from dilated bronchi.

Vessels.—The blood-vessels usually resist this process of destruction. They are to be found in the connective tissue, traversing the cavity. A section shows thickening of their coats as well as obliteration of their calibre. Often, however, aneurismal dilatations are to be found in the inner coat of large vessels, which may give rise to profuse and fatal hæmoptysis.

VII. *Interstitial sclerosis.*—In chronic tuberculosis, surrounding the infiltration, particularly in points which are destroyed and whose dissolution has not been too rapid, we observe a formation of neofibrous tissue, which may lead finally to retraction and solid cicatrices. Without recalling the fibrous tubercle of Grancher, we see too rarely this tendency to limit the process of caseation or ulceration by fibrous transformation.

Pulmonary induration.—These fibrous tracts form ordinarily pigmented brown or black indurations, but when

succeeding vast destructions of pulmonary tissue, they may reduce the lung to half its volume. The condensed fibrous tissues form, with the cavities and dilated bronchi, the anatomical substratum of those vast pulmonary contractions in which the cavities are ordinarily formed at the expense of pulmonary tissue, though more by the dilated bronchi, the dilation resulting from the traction of the cicatricial tissue on the bronchial walls. If cures are not more frequent, it is because from each tubercular focus the infectious matter escapes to attack new bronchi, so spreading the disease.

In the same way, tubercle, localized at the apex, gains little by little the inferior lobes, and the cough carries infecting material into the trachea so as to reach the other lung. When both lungs are attacked, vast injuries result, which are opposed to the function of respiration and compromise life.

VIII. *Bronchitis and catarrhal pneumonia.* — These inflammations are the result of the irritating properties of the bacillus, and perhaps also of the pus and other phlogogenous liquids, or from necrosed gangrenous particles, which may in their turn cause the development of an inflammation.

§ 19. *Nature of Tubercle in general.*

Bayle, who wrote ten years before Laënnec (1810), considered the granulation as the sign of a particular phthisis, distinct from tubercle. This opinion has been verified by Charles Robin, who has made the just observation that the granulation is not developed as tubercle; that it has a tendency to induration, to fibrous transformation; so that it stops before undergoing caseous degeneration. Sometimes, however, the granulation may soften in its centre. Virchow taught that granulation was the exclusive

type of tubercle, and that everything else was accidental and of inflammatory origin.

Bacillary granulation of the kidneys. — Benda has shown that in the kidney tubules the bacillus only determines desquamative nephritis, whilst in the glomerules of Malpighi, and around the capsule, it produces cellular induration, with inflammation of interstitial tissue and dilatation of glomerular artery, which is filled with bacilli. This is characteristic. The miliary granulation has no specific importance in acute phthisis. The bacillary focus is the true granulation of phthisis.

II. *Tubercle and pseudo-tubercle.* — These are distinguished by inoculation, the anatomical characters being similar. The tubercle of irritation does not easily produce an analagous lesion. It tends to localize itself. On the contrary, the bacillary tubercle is indefinitely inoculable and tends to diffusion.

Artificial giant cells.—Friedländer has seen these produced round foreign bodies in the lungs after section of recurrent nerves. Walb and Emile Marchand, after injecting the cornea with carmine solution, have seen giant cells developed, in the centre of which was found the colouring matter.

III. *Caseous pneumonia.* — Thaon, Grancher, Lepine, Wilson Fox, and especially Charcot, have furnished abundant proofs that caseous pneumonia does not exist without traces of tubercles or granulations. The dualism of Virchow thus loses its weight, and there is further this irrefutable demonstration, that the elements of caseous pneumonia are inoculable, exactly like the granulations and tubercles themselves, for the very simple reason that the bacillus dominates there. Virchow has, however, shown that caseous degeneration may take place, in all conditions of denutrition, as in the foci of suppuration, sarcoma, and typhoid

exudation. The dualist doctrine has its last refuge in the production of acute phthisis of inflammatory character. But this pneumonia is but the just manifestation of tuberculosis, to be recognized by the embryonic zone, the giant cells containing numerous bacilli. We find the proliferation of cells on the alveolar walls or in the connective tissue, and the same epithelioid cells in the cavity of the alveolus.

Conclusions.—1. The unity of phthisis, comprising all acute and chronic manifestations. There exists no dualism between tubercle and caseous pneumonia; no distinction between tubercular phthisis and inflammatory phthisis. 2. Analogy, even identity, of tubercular nodule, with inflammatory nodule from the histological point of view.

FOURTH PART.

Study of Causes.

VIII.

EXPERIMENTAL CAUSES.

§ 20. *Three Kinds of Experimental Phthisis.*

THE *first* is by insertion of virus under the skin, in the serous membranes, or in the anterior chamber of the eye. The *second,* by the artificial and forced use of food coming from the tissues of phthisical man, or from tubercular animals. Amongst these foods the one which most interests us is milk from tubercular cows. The *third,* by making our subject of experiment breathe air which has been contaminated and expired by patients, or, better still, air which has been in contact with tubercular products such as the expectoration dried and reduced to powder.

§ 21. *Inoculated Phthisis.*

Certain conditions are indispensable and necessary for the success of the operation.

1. The inoculated materials must be not only tubercular, but bacillary, and free from all septic micro-organisms.

2. The animal must be susceptible to tuberculosis.

3. The inoculation must be practised on organs or tissues which are not easily subject to inflammation.

I. *Materials containing bacilli.*—It has been discussed whether we ought by preference to use the substance of the miliary granulation, the tubercle, the caseous matter, or material from the so-called caseous pneumonia. With all these different substances the inoculation has been successful, as also with the *scrofulous* material from bone, glands, or the tubercles of the genital organs.

It has been shown that the bacillus occupies in the tubercles, whether local or general, the portions in a state of softening, the actual nodules, the central part of the neoplasm, and the giant cells; these, then, are the best agents for inoculation. Amongst liquids we must never trust to blood, urine, or excreted matters, but choose the pathological secretions of mucous membranes, the muco-pus in the products of expectoration, for these possess the maximum of virulence.

Fresh bacilliferous materials.—With the exception of the expectoration, which preserves its virulence for months, fresh materials must be used. The *débris* of tubercles in man, on account of the delay in autopsy, cannot be employed. Animals naturally or artificially tubercular cannot furnish any inoculable matters without being sacrificed. Putrid substances either give rise to septicæmia or produce no effect.

II. *Choice of Animals.*—Guinea-pigs and rabbits are animals very easy to inoculate or render tubercular, although little subject to the disease in a spontaneous form. Dogs, cats, and monkeys are also to be inoculated without difficulty.

§ 22. *Development of Inoculated Tubercle.*

Effects of inoculation.—This is practised in the subcutaneous cellular tissue, peritoneum, or, better still, in the anterior chamber of the eye. The inoculation is effected as

in vaccination, or by means of diluted solutions of tubercular matter with a hypodermic syringe. After some days local trouble at the place of operation is produced, but this does not affect general health.

Generalized tuberculosis.—At the end of a variable time the animal becomes weakened, wastes, and succumbs often after a colliquative diarrhœa, as with phthisical patients. At the autopsy we find the local tuberculosis already caseous, small miliary granulations round the inoculated point, tubercular engorgement of the glands, and granulations more or less caseified in the lungs, intestines, liver, spleen, kidneys, and peritoneal serous cavity. It is general tuberculosis.

Inoculability of artificial tuberculosis.—The tuberculosis thus produced is the true bacillary disease, for it can be again inoculated to animals of the same or different species, and that with certainty during several generations or successive series.

II. *Objections.*—In place of tubercular matter, for comparison, some observers have introduced into the peritoneum certain inert bodies, charpie, blotting-paper, or even fragments of cancer. They have injected into the veins either pus from an abscess, or the powder of lycopodium, and in all these cases have obtained besides local granulations generalized tuberculoid effects. Subcutaneous injection produces the same effect, and one sees by introducing an irritating powder, or an irritating liquid like croton oil, into the cellular tissue, that an anatomical process of the same kind as tuberculosis is produced. One may even see a coloured liquid appear in the giant cells which are developed at the inflamed positions. It seems, then, that tubercle does not act like a virus, that it constitutes but an ordinary irritant, and that the neoplasm which results resembles in all respects the tuberculoid forms obtained by irritant foreign bodies. From an anatomical point of view

this is incontestable, but if one considers the pathogenic character of the products, they are no longer the same. The ingenious experiments of Toussaint, and especially those of H. Martin, have definitely resolved the difficulty. When material from nodules caused by injection of irritant substances is used for inoculation, it *never* gives rise to general tuberculosis. It loses in the *second* term of the series of inoculations the power of producing even local inflammation. Thus the *specificity* of tubercle is demonstrated despite the anatomical similarity of the common lesions.

§ 23. *Inoculated Tuberculosis of the Eye.*

Cohnheim had the ingenious idea of introducing tubercular matter into the anterior chamber of the eye. The recent experiments of Baumgarten, styled "Demonstration of the Pathogenic value of the Bacillus Tuberculosis by Histology," leave no doubt as to the invasion and progressive attack of the bacillus in the different media of the eye; one sees, so to speak, the bacillus operate insidiously. The tuberculo-bacillary matter is introduced into the anterior chamber of the eye, and in the first four days no alteration or change is to be noted in the tissues of the eye; but each day sees the bacilli multiply *in situ*. Starting from the fifth day, the bacilli augmenting in number spread beyond the tubercular fragment to gain the iris and cornea. Then in the points where they abound and round them are produced new *epithelioid* cells, at first in small quantity, afterwards in increasing numbers, until a tubercular nodule is formed. The dimension of the tubercle and its richness in epithelioid cells correspond always to the quantity of bacilli. Facts of the same order are observed in the kidneys which develop bacilli; the parasites accumulate in the glomeruli, whilst the renal tissue is completely intact. It is to be noted that these experiments have been

made on rabbits, which constitute the most favourable cultivation ground for bacilli; in this animal tubercle prospers and always increases. It is not the same with the dog, which is but little subject to tuberculosis; he resists so well that inoculated tubercle ordinarily remains local and is arrested (Friedländer).

IX.

INOCULATED BACILLI.

As tubercle has been considered an ordinary non-virulent malady it has been necessary to make the bacillus itself the direct proof of inoculation. Koch (see second volume of *L'Institut Sanitaire*) has taken all the precautions necessary to render his experiments free from objection.

§ 24. *Planting Bacilli in the Eye, the Peritoneum, and the Blood.*

Bacilli have been cultivated on the coagulated serum of blood, collected afterwards with platinum wire previously burnt *clean*, so that the injection liquid was free from all trace of blood or other microphytes. It was composed exclusively of specific bacilli, which had been obtained sometimes from human tubercular matter, that is, a lung attacked with granulations or caseous pneumonia, sometimes from granulation material in the lung of the monkey, or from Lupus. The inoculation was made into the anterior chamber by injection through the cornea; in all cases, whatever the animal operated on, the same phenomena as in inoculation of tubercular matter were observed. With a liquid poor in bacilli a nodule was slowly produced in the iris, and little by little it infected the lymphatic

glands, which became caseous. Thence the morbid process passed by the blood, to propagate itself in other organs.

When the injection liquid is rich in bacilli the implantation tissue and the lymphatic system are rapidly overspread, and numerous nodules appear in the lungs, spleen, etc., as if the bacilli had been injected with the blood. Can we not explain by these differences in the march of the bacillary invasion why certain tubercles are arrested and definitely limited whilst in other cases the invasion is general and extremely rapid?

Bacillary injection into the peritoneum.—The same effects are observed according to the number of bacilli planted, when one operates with animals insusceptible or but little subject to tubercle, as dogs, rats, white mice. They do not die for several months, presenting at that time a very abundant eruption of tubercles in the viscera of the lower part of the abdomen, and but very few in the lungs. These facts, which are irrefutable, demonstrate the superiority of bacillary over tubercular inoculation. Here is one proof more—some rats were fed several months on tubercular materials without producing any effect; tubercular inoculations failed equally; but when the injection of the cultivated bacillus into the peritoneum was practised, a considerable crop of tubercles ensued.

Injection of bacilli into the veins.—When a very pure liquid containing bacilli only is injected into the blood itself, then miliary tuberculosis is more rapid and more general than that which is developed *spontaneously*.

X.

EXTERNAL CAUSES.

§ 25. *External Origin and Internal Development of the Bacillus.*

I. *Parasitic life.*—The bacillus tuberculosis develops in the serum of blood or in meat broth, but a temperature of 30° C. day and night is absolutely necessary. This temperature must be maintained for weeks, and if all these favourable conditions do not exist the bacilli are soon crowded out by other bacteria more rapidly prolific and less susceptible to changes of temperature. We have here an obstacle to the atmospheric development of tubercular bacilli; they are not true parasites which flourish outside the bodies of their living hosts. They do not multiply except in the animal body, but they produce spores which are spread in the atmosphere, and may enter into an organism so as again to produce bacilli. The atmosphere is not necessary for them to take the definite and durable spore form.

II. *Origin by metamorphosis.*—Their source from common microphytes passing through an infected medium has never been demonstrated by experiment. The bacillus tuberculosis once developed maintains indefinitely its miserable prerogatives; in culture liquids we have seen them intact for two years, and in the expectoration, even in the midst of putrefaction, the infecting qualities are preserved for six weeks or longer.

§ 26. *The Resistance of Bacilli.*

Falk (Berlin. Klin. Woch., 1883, No. 50) shows that the virulence of tubercular bacilli is much weakened by the process of putrefaction.

Baumgarten (Centralblatt, No. 2, 1884) relates experiments which appear to show that prolonged contact with substances undergoing putrefaction is necessary for the bacillus to lose its morbific properties. It preserves all other peculiarities including the colour reaction. The spores contained in the expectoration are very difficult to destroy, but boiling for twenty minutes is sufficient to render them harmless.

§ 27. *Tuberculosis of Respiratory Origin. Propagation by Inhaled Air containing Bacilli from Expectoration.*

I. *Conditions of penetration of lungs by bacilli.*—The respiration of air charged with the dust of dried *crachats* is the most certain form of transmitting human phthisis. The virulence of these dried *crachats* lasts for months, and depends on the development, more or less complete, of bacilli, and on the quality of spores they contain.

The handkerchief used by the patient is thus a very dangerous instrument for himself and others. When the bacilliferous dust is inhaled, it may, like other dust, be stopped in the upper respiratory tract, or pass into the alveoli, as when one breathes deeply with open mouth. The nose and larynx are serious obstacles to the entry of these matters, but they are also often expelled by the vibratile cilia of bronchial epithelium. As the bacilli are very slowly developed, a variety of favourable circumstances are necessary to locate them, such as stagnant mucus, adhesions making the lung unable to dilate completely, a vicious conformation of thorax producing the same effect as that of an accumulation of secretion in the bronchi, in which the bacillus is not only fixed but easily developed.

§ 28. *Bacilliferous Liquid Inhaled.*

Experiment by Koch.—The culture liquid to be inhaled was diluted, afterwards allowed to stand; a part subsided, the upper slightly muddy layer was decanted and 50 c.c. placed in a cupboard where eight rabbits, ten guinea-pigs, four rats, and four mice were shut up. From the fourteenth to the twenty-fifth day seven animals succumbed, the others were sacrificed on the twenty-eighth day. The guinea-pigs and rabbits had their lungs crammed with tubercles analogous to those obtained by inhalation of dried tubercular *crachats*, and which extended into the pulmonary alveoli like spontaneous tuberculosis. In the rats and mice the tubercles were not caseous as in the other animals. In all cases the tubercle artificially obtained when reinoculated to other animals provokes the development of general tuberculosis.

Conclusions.—Thus all these proceedings of bacillary infection by inhalation produce the same result as inoculation of the tubercular substance; they give rise to miliary tubercles, caseous tubercles, like the spontaneous disease; there is not even exception for insusceptible animals, as dogs, cats, etc. There are two hundred and eighty-seven experiments by Koch to prove a general law, the equivalence of bacillosis and tuberculosis. The bacillus is always found in true tubercles, never in pseudo-tuberculosis.

§ 29. *Penetration into the Blood. Consecutive Effects.*

Having once united into foci they may stop in their development; but in general they quit the primitive focus to penetrate the blood, either by effraction into the vessels, as Weigert has demonstrated, or by invasion of the lymphatic system, or even by erosion of the thoracic duct, of which Ponfick has cited a curious example.

Effects of microphytes in the blood.—When they have penetrated the blood in considerable quantity, or when they are found under conditions favourable for development, the blood is filled with products of decomposition, and becomes incapable of supporting oxidations: the animal dies. It is necessary also that the entry of these micro-organisms should be frequently renewed. The struggle between the blood and the parasites is incessant; the latter may succumb, producing in the blood chemical combinations which are hurtful to them.

Effects of micro-organisms on temperature. Causes of intermittent fever.—All these micro-organisms can elevate temperature; certain specific fevers are lit up, so to speak, immediately after the development of the parasites which give them birth. In relapsing fever one sees the strange phenomenon of its never appearing but with the entry of the micro-organism into the blood, and its disappearance with it. In malaria and tuberculosis the febrile movement *may* depend on the same causes.

Origin of heat. Oxidations.—Heat comes from decomposition of cells or from the process which the life of the micro-organism necessitates. It is unequally produced and distributed; at the highest point in the liver and spleen, at the lowest in the vena cava superior. It is to be supposed that in the diverse organs which produce heat there accumulate special and variable products of decomposition coming from the organs themselves, but which are mixed with different elements from the oxidation of micro-organisms.

Products of oxidation in the urine.—The urine nearly always contains aromatic compounds, which may be recognized by various colour reactions. The diabenzol often present indicates profound destructive assimilation of the body under the influence of micro-organisms.

XI.

ATMOSPHERIC ORIGIN OF MICRO-ORGANISMS.

The atmosphere is filled with spores of all kinds, it contains bacteria, more rarely bacilli; in every way it is necessary to determine the nature of these micro-organisms, if they are or are not specific.

§ 30. *Microphytic Atmospheres in general. Quantity of Micro-organisms.*

After the researches of Maddox in 1870, and those undertaken with the greatest success by Miquel since 1879 we know that the micro-organisms vary in quantity according to certain factors.

1. The season; maximum in summer, minimum in spring; but it must be noted that great heat for a long period diminishes the numbers.

2. Atmospheric pressure; the quantities increase directly as the pressure.

3. Dry seasons; these favour the germination of bacteria, which are rare in wet, rainy weather.

4. Ozone when in small quantity favours the germs.

5. Altitude has a very marked influence. The figures obtained by Miquel are—(*a*) Paris, 55,000 in ten cubic metres of air; (*b*) Montsouris, 7600; (*c*) Hotel Bellevue (Thoune, 560 metres), 25 in the external air, and 600 in a room; (*d*) Lake Thoune, 8. Above 2000 metres nothing was found. According to Miquel, this is caused by the dust being diminished and disseminated proportionally with the ascent, and also because the greatly rarefied air can no longer hold corpuscles in suspension. Above the snow-line no bacteria are found.

Methods of research.—According to Emmerich, all microphytes are developed more easily, energetically, quickly, and in greater number if we employ animal or vegetable liquids *sterilized by cold* (expressed meat juice or cabbage juice), than if we employ the same solutions deprived of parasites by boiling. It must be inferred that a principle very favourable to the growth of bacteria is destroyed by heating these liquids. The infectious inoculations undertaken by Fodor with the aid of bacteria taken from the air, and cultivated with isinglass solution, show that there are two pathogenic forms in the air (micro-bacterium agile and desmo-bacterium), and that they are capable of producing septic infections. The nutritive liquids are nearly always dominated by the ordinary bacteria of putrefaction. Emmerich has, it is true, excluded them for the advantage of other micro-organisms; but one cannot say that in so doing he has not destroyed pathogenic forms. At present our actual methods of research are imperfect.

§ 31. *Atmosphere charged with Particles containing Bacilli from Expectoration.*

Tappeiner in 1877 showed that dogs who were forced to breathe air containing pulverized materials from cavernous expectoration, all became affected with tuberculosis. His experiments have been contested, but also confirmed, by various observers.

XII.

ATMOSPHERIC CONDITIONS PRODUCING PHTHISIS.

These comprise—1. Life in towns contrasted with the country; the air of the former is eminently microphytic.

2. Overcrowding of human beings, as in barracks, shops,

schools, prisons; the air under such conditions is both confined and microphytic.

3. Climate exercises but little favourable influence, except (*a*) by the purity of the air, that is to say, the absence of germs; (*b*) by temperature, as far as it affects the multiplication of parasites; (*c*) altitude has the same indirect influence by producing rarity of micro-organisms.

§ 32. *Microphytic Atmosphere of Cities.*

Philadelphia registers 29 deaths per 100, from phthisis; Marseilles, 25; Paris, 25; London, 23·6; New York, 19; Stuttgart, 15; and Copenhagen, 13. Bouchardat has proved that those who have newly arrived in a great city suffer most heavily. The same experience holds in barracks; the mortality from phthisis is much more considerable amongst the conscripts who come from the country than with the acclimatized soldiers (Colin).

§ 33. *Hot and Cold Climates.*

Climate seems to have but a secondary influence; whatever be the latitude of the country, the mortality from phthisis is always greater in large centres of population. The countries or localities that remain free from phthisis have few relations with cities or with the commercial world.

§ 34. *Atmosphere of Altitudes.*

The evidence seems to be that they are beneficial only as free from the impurities of large towns.

§ 35. *Marine Climates.*

The air on the open sea is of extreme purity, but it must be remembered that this does not guarantee purity of

air on board ship. It has been asserted that the presence of malaria in a country has a protective influence against mortality from phthisis, but this is not borne out by inquiry into facts.

XIII.

EXTERNAL CAUSES.

Alimentary Phthisis.

It remains for us to show a third method of producing phthisis by feeding animals on tubercular materials, and amongst men by milk containing bacilli from tubercular animals or by the milk of a phthisical mother to her child.

Experiments on tubercular alimentation.—Chauveau in 1869 tuberculised three heifers by feeding them with caseous materials. At the autopsy he found enormous caseous masses, pulmonary tuberculosis, and glandular enlargements. We may say that well-conducted experiments by Gerlach, Chauveau, Klebs, Bollinger, Sommer, Orth, and Raymond agree in demonstrating the transmissibility of tubercle by *certain* aliments.

§ 36. *Alimentation by Tubercular Milk.*

When tubercular disease in the cow is confined to the lungs, and consequently localized, the milk is not dangerous; it becomes virulent if the tuberculosis is general. Boiling always destroys the virulence, even when the milk contains bacilli, which is the case when the udder of the affected cow is itself tuberculous. The question of suckling children by a phthisical mother has not yet been rigorously decided.

§ 37. *Tuberculosis of Different Origins.*

Penetration of respiratory organs by the bacillus.—In man it finds a home first at the apex of the lung, whence the rest of pulmonary parenchyma is often rapidly attacked, whilst the intra-bronchial infection from one lung to another is often wanting; nevertheless the expectoration may penetrate the other bronchi; in every way it is by progressive, direct transport of the bacillus, and not an infection.

Penetration by serous membranes.—Planting tubercles in the serous membranes of animals is followed by tuberculisation at first locally, afterwards of the rest of the organism. It is the same with man: tubercles of the pleura, pericardium, or peritoneum spread through the serous membrane with fatality and pronounced intensity.

Penetration of the digestive organs by the bacillus.—This is more difficult, as shown by the doubtful or often negative results obtained by feeding animals on tubercular matters. The gastric juice acts on the bacilli and destroys them by its digestive properties and acidity.

Intestinal tuberculosis in phthisical subjects.—The intestine is never tuberculised but when tubercular matters have often and for a long period passed into the digestive tube; in this case it is a true auto-infection by the intestine.

§ 38. *Of Phthisogenic Scales of Diet.*

Use of alkaline potash salts in excess.—Tubercular predisposition is no more than that there exist trophic modifications which favour the development of bacilli under certain individual conditions. Some individuals are preserved, although, living in the same air and hygienic conditions as those who succumb, they have only differences in their food. Phthisis and scrofula are to be seen in

the most diverse climates, under the most diverse hygienic conditions, in towns and countries, where the diet is the one thing arbitrary and variable; is it one of the causes of tuberculosis? If it is so, the poor may modify it as well as the rich. Chemistry tells us how much oxygen, nitrogen, and carbon are lost daily by the body, and the quantities of albuminates, fats, starch, etc., necessary to cover the deficit and sustain individual powers. We know that man, like certain animals, swallows an excess of hydro-carbons and indigestible cellulose, to obtain the feeble proportion of nitrogen contained therein. The digestive tract is thus surcharged with organic materials, but also absorbs a quantity of inorganic salts which may give rise to grave inconvenience. It is especially the alkaline salts which are in excess of the bodily requirements. The body of a dog, calculated for a kilogramme, contains in 100 parts of ashes, 8·49 of potash, 8·21 of soda, 34·84 of lime, 1·61 of magnesia, 0·34 of iron, 39·34 of phosphorus, 7·34 of chlorine. The body of the rabbit contains a little more potash than soda; the quantity of sodium chloride is the same in the carnivora as the herbivora. As all vegetables are rich in potash and poor in soda, it is the same with the milk of the herbivora. In meat, the food of carnivora, potash, and soda are in equilibrium, and they are found in the proportions required by the bodily expenditure of these animals. Calculating for an equivalent of soda we find—

In the whole body of carnivora ...	0·66 to 1·27 of potash.	
Total quantity in milk of carnivora	0·88 to 1·59	„
„ „ „ herbivora	0·76 „ 5·58	„
„ „ „ woman	1·33 „ 4·32	„
„ „ pure meat ...	3·98	„
„ „ cheese	12	„
„ „ rye	8·5	„
„ „ potatoes ...	31·42	„
„ „ peas	44·58	„
„ „ apples	100	„

Thus vegetable food, compared with human milk and the milk of herbivora, contains a considerable excess of potash. Meat is similar to milk in the quantity it contains. The *absolute* quantity of alkalies shows the same proportion; but, as demonstrated by Bunge in his excellent work on the relations of common salt and salts of potash in man (Dorpat, 1873), the quantity of soda in milk in general is but little in excess of that in all vegetables. The food of adult animals contains soda and chlorine in the same proportion for the herbivora as for the carnivora, whilst the quantity of potash in the food of the herbivora is from two to four times more considerable. To obtain the necessary supplies of albuminate for daily use they are obliged to consume enormous masses of vegetable matter containing much potash. That is perhaps why they are especially subject to tuberculosis.

The bovine race feeds on clover, hay, and grass rich in potash, and, like rabbits, hares, monkeys, and fowls, is much exposed to tubercular disease.

The horse and the pig are preserved up to a certain point by food containing much soda, as oats. The carnivora, dogs especially, rarely become tubercular, and are with difficulty inoculated. Rats and mice, however, have a greater resistance than the cat.

Effects of potash foods.—Although potash salts form an integral part of elementary cells and of connective tissue, they do not the less act as a paralyzing poison on the heart. Very diffusible, they pass from the intestine into the blood, and are eliminated by the urine. In this passage they extract the soda salts from the organism (Bunge), and the animals (as herbivora) which take food rich in potash salts must be provided with rock salt (NaCl) to maintain the normal proportion of chlorine and sodium, perhaps also for reasons more difficult to state precisely. A diet containing

potash salts is as necessary for the inferior organisms as the superior, and *we may suppose* that, in the herbivora, the tubercular bacilli increase easily on account of the presence of large quantities of potash salts in the body.

XIV.

PHTHISIS ACCIDENTALLY INOCULATED.

§ 39. *Bacillary Wounds.*

THESE have rarely provided true tuberculosis, probably for the reasons stated in respect to inoculation.

§ 40. *Vaccinal Tuberculosis.*

Conclusions.—(1) Vaccine lymph cultivated on tubercular subjects never contains bacilli, and consequently, never the tubercular infection. (2) The ordinary methods of vaccination are not capable of inoculating tuberculosis.

XV.

VITAL CAUSES.

§ 41. *Contagion in General.*

IN certain countries this is a popular tradition maintained in spite of the denial given by the most educated physicians. It has been the subject of several classical works, and there are many facts relating to its importation into regions previously free from such a scourge as tubercle.

§ 42. *Respiratory Contagion.*

Mode of transmission.—There are many well-established cases of matrimonial contagion (Compin, Weber, Mac Dowell), but it is probable that the mode of transmission was by

contamination of air with bronchial secretions. Genital contagion has not been demonstrated. The influence of habit or acclimatization in enabling us to resist respiratory contagion is marked. If we consult the documents furnished after twenty-five years' experience at the Brompton Hospital for Consumption by Williams (father and son), we shall see that the danger to the staff is not greater than under ordinary conditions. There were usually 600 patients, and 100 employés.

XVI.

MISTAKEN INHERITANCE. DIFFERENT PRECURSORY BACILLARY MALADIES.

Scrofula has passed as phthisogenic, but it is the same malady as tuberculosis itself. There are diseases which really are phthisis-producing—as, for example, diabetes.

§ 43. *Scrofula.*

Scrofula may be inoculated and reproduced as tubercle; scrofula contains in all its local forms the characteristic bacillus tuberculosus. Thus the two maladies follow each other; what more is wanted to prove they are identical? their relations are fixed.

I. *Scrofula inoculable.*—The experiments of Villemin, Cohnheim, Schüller, Kiener and Poulet, H. Martin, have proved that the products called scrofulous possess the same virulent properties as those of tuberculosis, and that in inoculating we determine local or general phthisis.

II. *Bacillary scrofula.*—All scrofulous lesions contain the bacillus. Ostitis, simple arthritis or fungoid arthritis (white tumours), are of bacillary nature. Adenitis, which must naturally be distinguished from syphilitic bubo, is

nineteen times out of twenty tubercular (Hérard). In the glands, as in fungoid arthritis, we find, not only miliary tubercle with the tubercular follicle, giant cells, and caseous conditions, but the bacillus, which is the essence of the disease. These local tuberculoses have nothing which differentiates them from tuberculosis in general; their characteristic (Féréol) is bacillary tubercle.

Testicular scrofula is nothing but local tuberculosis of the same origin.

Scrofulous lupus also presents signs of tuberculosis and contains the bacillus (Cornil).

The mild *scrofulides*, superficial affections of mucous membranes, certain coryzas, ophthalmias, and throat affections, seem to escape this identification, because probably not examined histologically. Quinquaud, in his excellent work on scrofula, makes certain reservations on the scrofulous constitution, the plethora of protoplasmic cells, that is, the swelling of tissues, and certain troubles in hæmatosis; he has nothing with which to constitute a distinct malady.

III. *Clinical combination of the two diseases.*—Quinquaud says that of tubercular patients $\frac{5}{12}$ have previously suffered from scrofula, or more exactly $\frac{1}{3}$ of the men, and $\frac{10}{17}$ of the women. It is, then, permissible to conclude that very frequently we find scrofulous antecedents in tubercular patients. The scrofulous ordinarily become phthisical at the age of puberty. Inversely, in seventy-four scrofulous children, at *Essmarck's clinique*, twenty-nine were found to have had phthisical parents. The scrofula of children is derived from phthisis of progenitors, as the phthisis of the adolescent from the scrofula of childhood.

§ 44. *Pretubercular Broncho-Pulmonary Lesions.*

Catarrhal bronchitis. Neglected colds.—These can no longer be admitted as causes of phthisis. It is the diffi-

culty of diagnosis at the beginning of the disease which has caused the popular error. When the commencement is by the larynx, the virus has determined ulcerous lesions, and it spreads to the bronchi, where tubercular nodules are formed, and which determine the *series* of colds, of which the *origin* has been neglected. What is true of laryngo-bronchitis is yet more true of catarrhal pneumonia. Generally the chronological order has been inverted. This is especially the case with regard to caseous chronic pneumonia, hæmoptysis, and pleurisy.

Chronic affections of bronchi.—Chronic catarrhs, bronchial dilatations, asthma, emphysema, far from being the cause of tuberculosis, may not only persist without provoking tuberculisation, but constitute true antagonisms. This is especially true of asthma.

Broncho-pulmonary affections of specific origin will be considered later (§ 47).

XVII.

VITAL CAUSES. RECOGNIZED INHERITANCE.

No individual circumstance plays such an important part in the development of phthisis as inheritance; but this must be made to comprise all forms of tuberculo-bacillosis, and not be limited only to the pulmonary form of phthisis.

(a) *Premonitory scrofula.*—If you learn that in a family there are grave, durable, articular, or osseous changes, suppurated or non-suppurated cervical adenitis, you may be certain that this is a form of scrofula, and, still more so, that the scrofula is only a form of tuberculosis. With a

like conviction you would not say that in passing from one generation to another scrofula is transformed into tuberculosis. It is tuberculosis itself under different forms. Scrofula is inverted tuberculosis.

(b) *Broncho-pulmonary premonitory signs.*—Hæmoptysis and pleurisy are to be specially noted.

(c) *Diabetes.*—It is not less important to take into account chronic maladies, which are not phthisical, but which precede or prepare the way for phthisis. I speak especially of diabetes. Between these two morbid conditions there is such connection that, in the same family, the diabetic sufferer gives birth to a tubercular offspring and reciprocally. People say that tuberculosis has but taken another form. Further, diabetes is often terminated by phthisis. In that case the bacillus is established in the lungs in an evident manner (Leyden).

§ 45. *Frequency of Hereditary Phthisis. Its Mode of Hereditary Transmission.*

I. *Frequency.*—Hérard and Cornil say 38 per cent. of the cases are hereditary. Mill and Cotton say half; and an extended inquiry by Professor Bockendahl, in Schleswig-Holstein, gave about the same result.

II. *Contagion in place of inheritance.*—It has been contended that many cases called hereditary are really examples of contagion. The child born into an infected atmosphere, and perhaps nourished with the milk of a tuberculous mother, acquires the disease by absorption of the bacillus after birth.

III. *Inheritance by direct transmission of tubercular virus. Analogies with, and differences from, the transmission of syphilitic virus.*—How can phthisis be due to a simple transmission of virus from parents to children? How is it that it does not act as syphilis, which is conveyed to

the child, even before birth, afterwards in characters visible on the body of the new-born infant, or perhaps is manifested in the first weeks or first year of life? Tuberculosis rarely attacks the fœtus, or new-born child. It is revealed in the early years, and in this respect approaches those tardy, hereditary forms of syphilis which have been so well described and demonstrated by Professor Fournier. It is a question in chronology that appears unanswerable at first sight.

IV. *Hereditary predisposition.*—To escape the difficulty it has been said that tubercular parents only transmit a *predisposition,* and not the disease itself. This tendency to contract tubercle will be increased by deficient nourishment, as milk from a phthisical mother. In my opinion it is a grave error.

V. *Scrofula in early life in place of phthisis.*—Phthisis is often manifested in early life under the form of scrofula, and this is what deceives. They have denied all transmissible characters to tuberculosis, at least such as those of syphilis, because one is precocious, the other tardy; but they forget a capital fact, which is that scrofula, demonstrated to be only a form of tuberculosis, has the domain of childhood. We see scrofulous glands, inflammations of joints, alterations in bone, grave forms of scrophulides, all affect children; but it is tuberculosis. There is only a difference in form; scrofula is peripheric, tuberculosis is visceral.

VI. *Meningeal and peritoneal tubercles in place of pulmonary phthisis.*—In an inverse sense we see pulmonary phthisis give place in infancy to tuberculisation of serous membranes, especially the meninges and the peritoneum. Meningitis and tubercular peritonitis may attack very young children, so that the objection of the slow evolution of tuberculosis falls before the fact, that in infancy it takes

the mask of scrofula, which is only an external tuberculosis, that later takes on the form of pulmonary phthisis.

Objections.—The gravest objection is this, that in the same family, collateral inheritance plays almost as great a part as direct inheritance. Several individuals may be spared, an entire generation may be passed over; and to explain this, moral conditions and hygienic circumstances have been appealed to.

Placento-fœtal blood.—This contains the germs of tuberculosis, and its inoculation produces the disease in the same way as if one had injected tubercular matter itself (Landouzy and Martin).

Consanguinity.—Between members of a family having the least scrofulo-tubercular taint, either remote or recent, marriage should be strictly avoided.

XVIII.

PATHOLOGICAL CAUSES.

THERE are maladies which favour the development of phthisis, others which appear really antagonistic to it.

§ 46. *Phthisogenic Maladies.*

We shall note: (1) a dystrophic malady, diabetes; (2) parasitic diseases, measles and whooping-cough; (3) professional diseases of lung, as anthracosis; (4) absolute starvation and complete ischæmia.

Frequency of diabetic phthisis.—The diabetic patient becomes tubercular forty-three times out of a hundred, and this malady is identical in all respects with true tuberculosis (Griesinger). This is so true that the bacillus has been found in the lungs of diabetic patients (Leyden), and especially in the *crachats* (see § 95).

Alternation of the two maladies.—The alternation between the two maladies in the same family constitutes an absolute demonstration of their reciprocal affinity.

Mode of tubercular development in diabetic patients.—Why and how are these people exposed to the virulent action of the bacillus ? There can be but two ideas as to this matter. Either glycohæmic blood is a favourable culture-liquid for the bacillus, or the diabetic patient becomes phthisical by inheritance, the phthisis not being revealed until an advanced period of the malady, when the individual is exhausted by loss of glucose. It is very difficult to fix the relative value of these two hypotheses.

§ 47A. *Influence of Microphytic Maladies on Bacillary Phthisis.*

I. *Measles.*—Do parasitic maladies exercise a favourable or unfavourable influence on the development of tuberculo-bacillosis ? There are incontestable differences to establish under this head. Typhoid fever, which otherwise attacks, especially the lymphoid tissues, is but rarely followed by tuberculosis; it is not the same with measles and whooping-cough, which exercise an undoubted phthisogenic power.

Frequency of rubeolic phthisis. — Whilst Rillet and Barthez indicate phthisis in the proportion of one case for every eleven of measles, Grisolle is doubtful, and Roger considers the complication a fact almost special to hospitals.

Mode of reproduction of rubeolic phthisis.—It is in reality, but in conditions of overcrowding air, vitiated by tubercle itself, that tuberculosis is most easily produced by way of contagion. It has been said that measles is only phthisogenic because it determines constantly a lesion of the bronchi with erosion and desquamation of epithelium, which facilitates the introduction of bacilli into the air-passages. We have already refuted this theory, which does not rest

on any foundation, seeing that rubeolic catarrh, generally slight and temporary, attacks only the upper mucous membranes, the bronchioles generally remaining free. It is, however, here that the bacillus is fixed when it becomes localized. When the malady determines broncho-pulmonary inflammation, this may conserve its character definitely—that is, until cure or death—without the pneumonia taking the caseous character, which is to say bacillary. The true cause of this affinity between measles and tubercle appears to be a double infection. Has not one, then, the right to ask if the microphytes which determine measles do not favour the development of bacillary spores? If there is antagonism, as we have demonstrated, between certain micro-organisms, there may also be identical conditions in their development. What is certain is that rubeolic phthisis, especially at the hospital, is ordinarily miliary, general and freely infectious; then the erosive lesion of the bronchi counts for nothing.

II. *Whooping-cough.*—Whooping-cough acts like measles as a phthisis-producing agent, but is more dangerous (Roger). We have equally to deal with a localized lesion in respiratory organs and a special micro-organism which presents, with that of measles and, without doubt, with that of tuberculosis, undiscussed relations. Here, again, one finds general tuberculosis.

III. *Syphilis.*—Syphilis plays an important part in the production of tuberculosis. Astruc and Van Swieten have already noted the fact. The syphilitic patient often dies tubercular. One finds then in the lung, syphilitic gummata by the side of tubercular granulations, which are very difficult to distinguish, and which cannot be recognized except by the presence of the bacillus. Further, the syphilitic sufferer often gives birth to a tubercular infant; is that on account of a cachetic state which is not constant or follow-

ing the transmission of one specific agent which favours the development of another?

§ 47B. *Professional Pulmonary Maladies.*

Respiration of foreign bodies, of dust, of different particles.—The professions which expose individuals to the respiration of air contaminated with irritating, mineral, animal, or vegetable dust, provoke pulmonary engorgements, metallic or mineral infiltrations, pneumonia of mechanical origin, rather than tuberculosis itself, which may, however, come at the end of these grave inflammations. Phthisis produced by inhalation of dust may be complicated with tubercular phthisis, especially when causes of contagion are to be found as in workshops (see the treatise on Hygiene, by Proust).

§ 48. *Influence of different Nutritive Conditions.*

I. *Inanition.*—True absolute inanition, especially that which results from a local obstacle to the passage of food into the stomach, is frequently phthisogenic.

Stricture of œsophagus.—Such especially as simple or cancerous strictures of œsophagus. Behier has already described the terrible consequences of this lesion of the alimentary canal, and numerous observations have confirmed his account. It is that, at the last degree of inanition, scarcely any oxygen is absorbed. It is the most favourable condition for the development of the bacillus.

Persistent anorexia.—It is the same with invincible anorexia sometimes met with in hysterical subjects; with little oxygen in blood and tissues, plenty of bacilli are to be found.

Ulcers of the stomach.—These have also been described as phthisogenic, though only by error or false interpretation. It often happens that young phthisical patients, especially

women, present solitary or numerous simple ulcerations of the stomach, as one observes in common conditions of debilitation. There is no relation of cause and effect; it is a simple coincidence. For stronger reasons, we must consider it an error to say that tuberculosis is developed as a consequence of gastric ulcers.

Ischæmia. Blood inanition.—All congenital maladies of the heart, particularly contraction of pulmonary artery, have, as a consequence, the development of tuberculosis. This is clearly shown by the collected facts of Lebert, Constantin, Paul, and others. We may admit that impaired circulation of blood in the lungs is a favourable condition for the development of the bacillus.

Aneurism of aorta.—I have observed three facts, clearly proving that aortic aneurism may provoke the development of tubercles in the right lung, corresponding to the arch of the aorta. There is here an analogous trouble in the circulation, a sort of static ischæmia, which produces the development of the bacillus.

Anæmia.—By the same process of reasoning the theory of inanition has been applied to abnormal conditions of the blood attributing to anæmia a tuberculising power. There is only a false analogy, and we often see the most serious anæmia without its producing tuberculisation.

Chlorosis.—Chlorosis is often exclusive even of phthisis. I know tubercular families in which the chlorotic members have been preserved from phthisis. What leads to theoretic error is that sometimes chlorosis simulates phthisis in a complete manner. On the other hand phthisis, at its commencement, is often shown, but by a pseudo-chlorotic state, which, in its turn, is difficult to distinguish from chlorosis (see *Traités des Anémies*, 1867; see also § 55).

49. *Pathological Antagonism. Immunities.*

Scarlatina seems to be really exclusive of tubercle; but small-pox, typhoid fever, and malaria are not admitted as possessing antagonistic powers. Neither alcoholism nor cancer appear to exercise any preservative influence.

Dystrophia. Arteritis.—In the cachectic period tuberculosis may terminate any of these conditions. Whilst the gouty patient has nothing to fear during the acute stage, when the malady becomes chronic it may degenerate into phthisis (Pidoux). Phthisis, says Pidoux, is a malady which *finishes*. Peter judiciously rectifies this word; it is a malady which *despatches*.

Heart diseases.—The school of Rokitanski have thought that there is a dyscrasic antagonism of blood between maladies of circulation and tuberculosis. Cyanosis, especially, should be opposed to the production of phthisis; it is precisely the reverse, at least in congenital maladies of the heart. For acquired diseases of the valves, eight per cent. of the patients have been found also tubercular.

Asthma. Emphysema—We intend to speak especially of asthma—properly so called—which is accompanied always by neuro-paralytic emphysema (paralysis of vagus nerve). It may be temporary or permanent; in these cases the lung, losing its elasticity, becomes like a damaged, softened india-rubber bag, and no longer acts for inspiration because of the rupture and confluence of its air vesicles. The microphyte can scarcely enter or develop in this inert sac.

§ 50. *Experimental Immunities.*

If successive cultivations of the bacillus are made in hermetically sealed vessels, it will preserve its virulent powers indefinitely; but if the vessels are badly closed, the later generations will have diminished virulent properties. Falk

has attempted inoculating with tubercular virus attenuated by heat, but without results. Pasteur has formulated the following law: — "When an animal has been inoculated with a diluted poison, and the organism has overcome it by its resistance, an inoculation with the condensed poison produces but insignificant effects." The poison may be understood especially in connection with micro-organisms.

XIX.

PHYSIOLOGICAL CAUSES.

§ 51. *Individual Conditions of Health, Physiological Misery.*

Predispositions.—Predisposition, as we shall see, is the unequal facility of different individuals in submitting to bacillary infection. To explain these differences of liability to contagious maladies, feeble conditions, either inherited or acquired, have been particularly invoked. Bouchardat has invented the ingenious term *physiological misery* to designate the assembly of various unfavourable circumstances. Insufficient nutrition, insufficient aeration, debility from exhausting conditions (such as overwork, prolonged diarrhœa, chronic dysentery, seminal losses, too-long continued lactation), have all been accused of producing this *predisposition* to tuberculosis. One sees people exposed to all these unfavourable circumstances escape; whilst the most robust individuals, in favourable conditions of fortune and hygiene, fall, as if struck by lightning. The explanation is in these two words: contagion and inheritance.

§ 52. *Physiological Causes. Influence of Age and Sex. Frequency of the Malady.*

Frequency. Proportion of deaths by phthisis in general

mortality.—In the populations of cities one counts generally an annual mortality of thirty-four in ten thousand inhabitants; the extreme limits are twenty-five to fifty-three. Relatively to the general mortality, the proportion from phthisis is one-fifth, or a fourth part; thus, at Paris, in a thousand deaths weekly, we count at least two hundred from pulmonary phthisis, and further twenty or thirty deaths from other forms of tuberculosis, which makes the total twenty to twenty-five per cent. In medico-legal autopsies, which are made on individuals reputed to be healthy, Professor Brouardel has found the enormous proportion of four-fifths with tubercular lesions in all stages.

Age.—The distribution of ages as to tuberculisation is difficult to appreciate; M. Damaschino, in his excellent *thèse d'agrégation*, said very judiciously, twelve years ago, that we must count *all.* tubercular maladies. This is especially true of children who commence so often by tuberculosis of other organs than the lung.

Fœtus and newly born child.—From time to time, in autopsies of the fœtus and of newly born children, masses, called tubercular, have been described as existing in the lungs, or in the glands. This is from caseous pneumonia or adenitis. Are the tubercular masses bacillary *or not?*

From birth to three or four years.—In children aged less than one year, Hervieux has found tuberculosis ten times in eight hundred and one autopsies.

From three to eight years.—Starting from the age of three years up to seven or eight, the malady makes such an increase that in hospitals for children two-thirds of the deaths are caused by phthisis (Rilliet and Barthez). Tubercles are often found then in the meninges and the brain (Roger).

From eight to fifteen years.—There is a decrease noticed by all authorities. It is true that at this epoch other

bacillary forms predominate; scrofula under all its forms, ganglio-bronchial tuberculisations, etc.

Pulmonary phthisis more often takes the form of caseous pneumonia at this age, and more rarely that of granulation than in first childhood.

From fifteen to twenty-five years.—This age is said to be the maximum for frequency.

From twenty-five to thirty years.—The extreme limits of maximum mortality are from twenty-five to thirty-five to forty, and even to fifty years.

Old Age.—It is not rare even in old age, as shown by the autopsies made at the asylums for old people. What often deceives the practitioner is the insidious progress of the disease, which developes without, so to speak, troubling the pulmonary functions, without giving certain physical signs, and without provoking fever. This, then, is latent phthisis in every acceptation of the word.

Conclusions.—No age is exempt from the disease; and if its maximum is between fifteen and forty-five years, it is between these limits that the occasions of contagion are most implied, most frequent.

Statistics.—The following are exact figures given by Bertillon for the annual mortality in the city of Paris, during the years 1872 and 1877, the total population being classed in series according to age.

Below one year ...	...	163 deaths for	141,162 children
From 5 to 10 years	...	59 „	127,357 „
„ 10 „ 15 „	...	125 „	133,662 „
„ 15 „ 20 „	...	557 „	168,000 individuals
„ 20 „ 25 „	...	989 „	222,000 „
„ 25 „ 30 „	...	1189 „	200,000 „
„ 30 „ 40 „	...	1228 to 1213	188,000 „
„ 40 „ 50 „	...	750 „	145,000 „
„ 50 „ 55 „	...	480 „	110,000 „
„ 55 „ 60 „	...	295 „	77,000 „
„ 60 „ 65 „	...	181 „	58,000 „
„ 65 „ 70 „	...	103 „	36,000 „
„ 70 „ 75 „	...	73 „	23,000 „

Sex.—Starting from the beginning of sexual life, the females have a morbid predominance. Fuller states their mortality to be twelve per cent. greater than that of men.

Pregnancy. Puerperal conditions.—Pregnancy aids powerfully in the development of phthisis; and it is only by masking symptoms that pregnancy can appear to arrest its progress. The puerperal state starts the process of tuberculisation into singular activity; further, after the confinement, tuberculisation often attacks the genital organs (Namias, Cruveilhier, Brouardel). Is it a result of contamination from the phthisical husband?

Prolonged lactation, from its debilitating effect, is injurious. Phthisis is more often inherited by women than by men.

XX.

LOCAL CONDITIONS.

§ 53. *Sedentary Life. Conformation of the Chest.*

Respiratory inertia. Sedentary professions.—Want of exercise is considered by Clark as one of the most fatal circumstances. It appears certain that deficiency in aeration of the lung favours the arrest and multiplication of the bacillus.

Vicious or paralytic conformation of the chest.—Contraction of apices of the thorax, diminution in their circumference (Hirtz), projection of scapulæ, prominence of ribs, these are the signs which constitute a warning, or even have the value of an accomplished fact. It is the same with excessive shortness of the three highest ribs (Freund) and the weakness of the thorax, known under the name of paralytic thorax; it has the effect of diminishing the vital

capacity of the chest. These, then, are causes or effects; causes in the sense that non-performance of functions by the upper portions of chest fixes the bacilli, as we shall demonstrate in the following chapter; they are effects in the sense that old tuberculosis, usually accompanied by pleurisy, weakens and flattens the chest in all its diameters.

§. 54A. *Penetration of Bacilli into the Apices of the Lungs. Therapeutic consequences.*

I. *Anatomical facts. Localization of tubercles in the upper lobes of the lungs.*—All practitioners agree with the anatomo-pathologists as to the order which reigns in the distribution of tubercular lesions, and recognize, in long-standing phthisis, the most significant lesions in the apices of the lungs. Often they remain limited at the apex or its neighbourhood; and when they spread or multiply, it is by proceeding from the superior to the inferior lobes.

II. *Explanations.*—Several explanations have been given of this undoubted fact.

(*a*) *Conformation of the chest.*—Waldenburg and Freund have attributed it to the conformation of the chest, that is to say, to its incomplete inspiratory distension at the upper segments, from which an accumulation of mucus and epithelium in the bronchioles results. The idea is that the upper portions of the musculo-osseous chest wall do not expand properly during inspiration.

Functional inertia.—Jaccoud admits functional inertia of the superior lobes as a cause of the localization of tubercles, and counsels respiratory gymnastics as a preservative measure.

Functional degradation of the pulmonary apices.—Peter says that in the lung, which is so poor "as to texture, and so passive as to function, the apex is precisely the least living part, for it is the least acting." Again, in the apex

the air arrives and circulates with most difficulty; it is there, consequently, where we find the minimum of hæmatosis, the maximum of tuberculosis, which is itself a trophic deviation, that is, the contrary of inflammation.

III. *Interpretation of the localization of tubercle in the apices.*—There is direct penetration of bacilli into the upper bronchi. The bacilli, or the spores, which may in germinating form parasites, are transported into the bronchial trunk by the current of inspired air; they penetrate more easily into the right bronchus, which is the larger. In hereditary phthisis, the left lung is usually affected (Lancereaux). Once arrived in the smaller bronchi the micro-organism finds in the upper lobes special conditions of development; their phthisogenic monopoly is easily explained by innate feebleness and little use of these lobes in the functions of respiration. On account of the fixity of the superior ribs, the relative inability of the inspiratory muscles (intercostals, upper portion of pectoralis major and minor), these lobes expand much less on inspiration than the middle and inferior portions of the thorax, of which the mobility and muscular equipment is very differently developed. What is still better marked is the imperfection of the expiratory act, which, in the physiological state, is due only to contraction of the elastic portion of the ribs. These first ribs are still more unable to contract on themselves than they are to expand. On account of the last condition, the air is incompletely expelled. We know that the air which remains in the lungs, residual air, after the respiratory act, is nine-tenths; there is hardly one-tenth renewed. The ventilation is especially incomplete in the upper regions of the thorax; there is stagnation of air, stagnation of mucosities. The micro-organism remains there without being disturbed or carried away by the movements of the air. It is only when the lung is emphysematous, without elas-

ticity, and absolutely inert, that the bacillus is neither fixed nor developed.

The development and multiplication of bacilli is effected in the apices of the lungs. — The special conditions of development are—(*a*) Absence or diminution of oxygen; (*b*) increased temperature, stagnation of mucus.

Therapeutic consequences relative to medicinal relations. —If the development of tubercles follows step by step the introduction, fixation, and multiplication of bacilli in these fatal regions, it is there we must try to act; if an artificial atmosphere, if antiparasitic or antiseptic vapours can reach the bacilli confined primitively in the bronchioles, we may hope to prevent their indefinite multiplication, and transform the malady into a local circumscribed tuberculosis.

The question of local tuberculosis is in reality now decided for all the organs, bones, glands, etc.; why, then, should not the parasitic invasion be limited to a portion of the respiratory organ?

FIFTH PART.

Clinical.

From a semeiological and diagnostic point of view, that is to say, from that of actual practice, phthisis presents itself under four forms.

First category. Latent phthisis.—It remains latent by physical signs, by functional symptoms, and is only revealed by a micro-chemical examination of expectoration.

Second category. Distinct phthisis.—Distinct phthisis is recognized by auscultation and percussion, but it is often confirmed only by an examination of expectorated matters.

Third category. Masked phthisis.—This category comprises phthisis which takes the mask (*a*) of another pulmonary disease (bronchitis, pneumonia, congestion, emphysema); (*b*) or of an extra-pulmonary thoracic affection (laryngitis, pleurisy, fever, and circulatory troubles); and, lastly (*c*), of an extra-thoracic lesion, as genito-urinary or intestinal tuberculosis.

Fourth category. True and false cavernous phthisis.—The last group, which comprises phthisis arrived at complete development, presents, nevertheless, a doubtful semeiology. In other words, although characterized by the positive signs of induration and pulmonary excavation, it often resembles indurations of another nature—tumours, and simple bronchial cavities. Reciprocally the same maladies may simulate extensive and cavernous phthisis; this double cause of error will justify the terms true and false cavernous phthisis.

FIRST CATEGORY.

Latent Phthisis.

XXI.

DIFFERENT FORMS.

Very often phthisis is manifested only by phenomena of denutrition (chlorosis, dyspepsia, loss of flesh, fever), or by certain functional troubles of the lungs (cough, hæmoptysis, etc). Auscultation is uncertain in the initial period; certainty can only be acquired by the micro-chemical method.

§ 54B. *Latent Denutritive Phthisis.*

Denutrition is one of the initial marks of tuberculosis; chlorosis or anæmia, with or without menstrual troubles; dyspepsia, with or without vomiting; night-sweats, loss of flesh, fever; here are the first signs of bacillary infection which seems to act on all the organism before provoking local manifestations.

I. *Chlorosis.*—Very often phthisis is only represented by the appearances of chloro-anæmia, especially amongst women, without it being possible to invoke either hygienic conditions, bodily formation, or marriage, which has no part whatever in the production of the mischief. Thus, without appreciable motive, the subjects become pale, a dull yellow paleness of an earthy tint, which differs totally from the greenish colouration of true chlorosis. They are very easily fatigued, and complain of a painful lassitude in the limbs, which symptoms exist only at a late period in chlorosis.

Palpitations are manifested at the least moral emotion or most trifling muscular exertion. In the intervals of palpitations, the action of the heart is nearly always accelerated, even during repose, but more particularly in the evening.

The *sounds of the heart* are accompanied by a metallic click, rarely by a well-characterized murmur, and more rarely still by a continued murmur in the vessels of the neck which constitutes the rule in chloro-anæmia.

Respiration is often difficult, in going upstairs almost impossible, whilst it is calmed at night, which is not usually the case in tubercular disease.

II. *Menstrual troubles.*—What attacks and torments women most is menstrual perturbation, which often constitutes an initial phenomenon. The monthly loss of blood becomes less abundant and irregular, it may end even in total suppression, although less tardily than in chlorosis. With young girls at the period of puberty, latent tuberculosis ordinarily hinders the establishment of menstruation; in all cases it is difficult and incomplete. In chlorosis, amenorrhœa is often complete from the commencement of the malady, which is manifested and expressed precisely by the suppression or absence of menstruation. Thus, there are great analogies, but also sufficient differences, to enable us to avoid an error which is possible and frequent. After having exhausted the parallel, pay attention to the little dry cough, with or without paroxysms, with or without vomiting. The decisive phenomena have to be described. I wish to speak especially of digestive troubles, loss of flesh, night-sweats, and the evening elevation of temperature.

III. *Primordial dyspepsia.*—Tuberculosis is yet more often marked by dyspepsia than by anæmia as a premonitory sign. Bourdon has noted digestive troubles in more than two-

thirds of the cases of commencing tuberculosis; often it is manifested before there is the least physical sign or the least functional change in respiration. I make exception but for the cough which precedes dyspepsia, especially the cough accompanied by vomiting. This vomiting, which at first sight appears to depend on the state of the stomach, should not be classed amongst the number of dyspeptic accidents. It is the mechanical effect of the cough, the stomach performing its function as in the normal condition during the interval of painful paroxysms. Dyspepsia is manifested in the great majority of cases (see "*La dyspepsia tuberculeuse*" in our "*Traite des dyspepsies*") by loss of appetite, which is not recovered, except with the aid of stimulating food; by eructation of gas, indicating decomposition from digestive troubles. The epigastric region is raised, and becomes painful on pressure or the ingestion of food. The evacuations are rare, and if the vomiting provoked by the cough comes to complicate the dyspepsia, the mischief makes rapid progress, although an autopsy reveals but insignificant lesions, or, at most, a certain degree of inflammation.

IV. *Loss of flesh.*—In phthisis of chronic and apyretic character, the nutrition of the patient may remain for a long time in the normal state; but it is not so in tuberculosis which starts suddenly, or which follows an acute course. From the first beginning of the illness, before even the appearance of pulmonary phenomena, the patient notices a rapid wasting, often a real colliquation of fatty and muscular tissue, particularly in the chest.

Causes.—(*a*) Where the appetite is lost, the digestion troubled, and food rejected on account of the cough, inanition is quickly established, and wasting is the inevitable consequence. (*b*) If the commencement is marked by fever, that is by consumption of tissues and loss of albuminoid

materials, mischief is yet more prompt and inevitable. (*d*) Most frequently the loss of flesh takes place without apparent cause, without previous conditions, without known moral influence. Beware of latent phthisis; when accompanied by a little dry cough there is danger.

V. *Sweating.*—The appearance of profuse sweats, especially at night, which come to join the loss of flesh, and the cough, constitutes a new cause of alarm without one knowing either the mode of production or the cause of the diaphoresis which is absolutely independent of fever. Habitually we have been content to attribute these sweats to weakness, which is not an explanation, seeing that they are often initial. When they coincide with fever they are manifested, especially during the febrile remission, in the second part of the night. The sweats have been explained by an accumulation of CO_2 in the blood, or rather, in the centre of sudation (Lauder Brunton).

§ 55. *Diagnosis of Latent Chloro-anæmic Phthisis.*

Latent chloro-anæmic phthisis.—When phthisis is manifested with the signs of chloro-anæmia it can be characterized by a single word—it is a general dystrophy; and whilst the chloro-anæmia bears *principally* on the blood, all the organs, tissues, all the liquids suffer as a consequence, because tuberculosis has taken possession of the organism. The blood is attacked in the same degree and in the same proportion as the other elements of the economy. We have a double proof of it. At first in comparing phthisis and chloro-anæmia there is a single point in common, that is, the *alteration of the blood*, and the series of phenomena which result from it, we can be assured of their predominance in primitive chloro-anæmia and of their minor significance in organic chlorosis.

1. The bellows-like sounds in great vessels which are so

marked, so characteristic of the diminution in number of the red corpuscles, and of their alteration in form, is wholly wanting in organic chlorosis. 2. The cardiac *souffle* at the arterial and auriculo-ventricular orifices is never a true murmur: it is rather an exaggerated clacking sound. 3. The circulation is constantly accelerated in anæmia-producing tuberculosis, and the palpitations never fail, although they may or may not be complained of by the patient; with chlorotics the circulation is ordinarily of the normal type. 4. Lastly, the dyspnœa of chlorotics is much more tardy and complete because of the diminution in hæmoglobin of the globules, which is the respiratory agent. There are, however, positive proofs of the denutrition which immediately attacks the phthisical patient. (*a*) Muscular fatigue is early and complete. It is that which hinders walking, especially on a rising ground, which requires more muscular effort; the muscles appear to suffer even at the outset from denutrition; dyspnœa, which seems also to be of the muscular order, is produced by the least movement both at the beginning of the illness, and in a permanent manner. (*b*) The skin, instead of being discoloured, presents a dull, greyish tint as in cachexia. (*c*) Chlorosis is apyretic, or rather it never presents a true febrile movement like tuberculosis. (*d*) Wasting is marked from the beginning, whilst ordinarily the condition of the chlorotic patient is preserved. In conclusion, the state of the muscles, the skin, general nutrition and oxidation all indicate immediate damage, which, with the chlorotic, affects only the blood.

§ 56. *Latent Dyspeptic Phthisis.*

The tubercular patient often commences his malady by a dyspepsia, which must be carefully distinguished from simple dyspepsia, whether followed or not by loss of flesh. In the second place, pretubercular chlorosis gives rise to

digestive troubles, which border equally on primitive dyspepsia. We shall have afterwards to speak of a phenomenon of which the origin has often been sought in the stomach, the gastric cough. Then it will be useful to indicate gastro-intestinal troubles which are under the influence of tænia or ascarides, and which in children especially are developed often to the point of simulating phthisis. The minute distinctions which I seek to introduce into diagnosis may doubtless appear useless or exaggerated; to justify myself, I appeal to the memory of the practitioners who have had to grapple with difficulties.

Phthisis of dyspeptic form.—Abstraction having been made as to vomiting, which is of mechanical origin, we shall state as indices of the dyspeptic form of phthisis, complete absolute anorexia which applies ordinarily to all food; slow, painful digestion, with a burning sensation along the œsophagus, pyrosis at the pit of the stomach, acid regurgitations, gaseous eructations containing the gas from fermentation of the alimentary mass, dorsal and gastric pains, tympanic swelling of digestive organs with constipation, more rarely diarrhœa, at least at the commencement. The picture is rarely so dark, abnormal sensations are not always united and associated in totality, with the phenomena of properly called chemical decomposition; but it is sufficient that it may be so to have the right of putting this morbid series in parallel with those which start from primitive dyspepsia. It is to be noted that the latter is never so absolutely characteristic, nor so apeptic, nor so durable as pretubercular dyspepsia.

It must be thus that the evil is less profound in the dyspeptic than the phthisical patient, for he does not lose so much of his strength, fat, and muscle, nor so rapidly and completely as the tubercular sufferer. So that here again we can say that the denutrition which affects all tissues, all

organs, takes in the development of emaciation a part as great as inanition by default of assimilation or alimentation. Exaggerated oxidation, insufficient repair, are the double causes of failure, whilst in the dyspeptic there is but defective assimilation.

Phthisis at once chloro-anæmic and dyspeptic.—In pretubercular phthisis it is rare that the digestive functions remain in the normal state; in simple chloro-anæmia these are rather nervo-motor troubles than chemical dyspepsia. In anæmia of organic origin it is the inverse, as we are about to demonstrate. The chlorotic has a capricious appetite, but not an absolute want of desire for food. Such patients have gaseous distension, but the gas is from atmospheric air introduced into the stomach at the moment of deglutition of food, and not from the gas of fermentation. The pains are of a physico-nervous order, and arise from the distension of the muscular coat of the stomach by inert gas. For the rest, what proves they are not of a chemical order is that the assimilation of aliments transformed into peptones continues to operate as in the normal condition, and the invalids do not lose flesh; it is the inverse in the tuberculo-gastric condition.

Gastric cough.—Stomach cough is yet keenly discussed. Physiology cannot reproduce it; clinical medicine is more explicit. If this cough exists it must be distinguished from the cough of tubercular disease.

Cough produced by worms.—This is accompanied by a series of nervous phenomena which recall hysterical symptoms; by itself it presents no special character, although it appears more real than gastric cough. The expulsion of the worms ought to stop it, "whilst nothing arrests the famous stomach cough, either in tuberculosis where it does not exist, or in nervous states where it must be distrusted."

§ 57. *Latent Febrile Phthisis.*

I. *Tubercular fever.*—Perhaps the initial fever is manifested by a slight elevation of temperature, with acceleration of pulse, without being preceded by true shivering, or followed by perspiration. It starts in a third of the cases with the first local symptoms, and even before them; in another fifth of the cases it is manifested in the course of the first period (Louis). According to Sydney Ringer, the elevation of temperature is continuous, but also little marked, and the thermometer seldom registers more than half a degree, or a degree (centigrade) above the normal, at least, unless there comes a recrudescence in the tubercular eruption. Generally it appears more marked towards evening, and this exacerbation coincides with the evening elevation which takes place normally. At this moment it is rather characterized by acceleration of the pulse, which makes it appear more intense than it is in reality. The application of the thermometer permits us to appreciate exactly the degree of the fever, and to distinguish this state from chlorosis, which but rarely provokes a like excitement of the heart, and never hyperthermia.

There is a danger to be avoided, for, if we treat this pseudo-chlorosis, by causing erethism of the circulation with the aid of iron, we may be quite sure to augment the gravity of it, and give an injurious impulse to the latent lesions of tubercle.

II. *Intermittent fever. Diagnosis.*—It is not rare to see tuberculosis at its commencement simulate intermittent fevers. I have seen several facts of this kind. A lady attended by a hospital physician was sent for sea-bathing on account of a very obstinate intermittent fever. She set out in spite of my counsels and my diagnosis. At the third bath she had hæmoptysis, and soon after all the signs

of acute caseous pneumonia. The thermometric examination of phthisical subjects shows that the elevation of temperature is never in the morning. It is never freely intermittent, and is never elevated as in malarious fevers. Sulphate of quinine has no effect, and the spleen is not affected. If actually a case of this kind came under my observation, the presence of a *crachat* would be sufficient for me to make the diagnosis by finding the bacillus.

III. *Vaso-motor troubles.* — The vaso-motor nervous system is singularly excited from the commencement; at the least physical or moral impression, the face flushes and the cheeks become injected either on the affected side or on both.

IV. *Nervous troubles.*—The nervous cerebro-spinal system is often troubled from the beginning; the invalids, especially when they are in a state of febrile or pseudo-febrile erethism, become hypochondriac, melancholic. Sensibility is increased, especially in women who have already shown signs of hysteria, although the intelligence remains perfectly intact, and that during all the course of the malady. The moral agitation, vaso-motor excitement and acceleration of the heart, constitute a remarkable collection of phenomena which often finds its true signification in slight vesperal hyperthermia, and which in every way establishes very clear distinctions from the chloro-anæmic state.

XXII.

SEMEIOLOGY OF LATENT LOCALIZED PHTHISIS.

Pulmonary phthisis is tuberculo-bacillosis localized in the lungs, whilst miliary phthisis invades all the organism. At present there is no discussion as to whether there are several kinds of phthisis. There are not six species, as

Bayle wished to establish, nor two, tubercular and inflammatory, as certain schools of German anatomo-pathologists still desire to prove. The differences between the so-called species of diathetical phthisis (that is to say arthritic, scrofular, etc.) do not exist, as some French theorists still maintain. It is always tubercular as taught by Laënnec; I add always bacillary as demonstrated by Koch.

I. *Variations of phthisis.*—Despite its unity, it varies in its forms, its course, periods, and manifestations, but it is always the same phthisis. Under this heading we must distinguish—

1. *Ordinary phthisis,* which presents three periods, the bronchial, the pneumo-caseous phase, the ulcero-cavernous state with or without sclero-fibrous transformation.

2. *Subacute phthisis,* or acute or galloping, which may be developed in a few weeks, following the same stages, and producing the same lesions as ordinary phthisis, from which it does not differ except by the rapidity of its course; it does not merit a separate description.

3. *Granular phthisis,* or rather granular tuberculosis, which alone requires a categoric distinction because it ends infallibly in death, although resting ordinarily granular without passing through the ulcero-cavernous periods.

II. *Bacillosis and premonitory maladies.*—In the great majority of cases, pulmonary phthisis is primordial; but it is not always so; there may be preliminary bacillosis which is forgotten on account of its silence, concentration, and cicatricial transformation. Recollection of this should be in the mind of the practitioner, who endeavours to recognize latent phthisis in the midst of initial manifestations, often strange and differing in appearance, like chlorosis, fever, dyspepsia, sometimes perfidious as hæmoptysis, or cough, or, lastly, with the double meaning of certain signs of auscultation.

III. *Extra-pulmonary bacillosis.* — Phthisis is latent when one does not find certain characteristic signs in the lung. It is masked, that is to say, hidden by other maladies, when it commences by ulceration of the larynx, which is frequent, by tuberculosis of the pleural serous membrane, by specific peritonitis, by affections of the genito-urinary organs, which are cured if often to be reproduced later; lastly, by manifestations called scrofulous, but really tuberculo-bacillary in bones, articulations, and glands. We must not despise these grave warnings, which may be slight, remote, or neglected, but which nevertheless demand attention from the observer.

IV. *Enumeration and mode of appreciation of different symptoms of phthisis.*—In studying the symptoms whose union constitutes phthisis, we have two dangers to avoid and one precaution to take. 1. To avoid the general description of a sign with respect to any malady whatever; only the mechanism of functional or physical trouble interests the practitioner. 2. In the malady which occupies us, we have not to give the history of symptoms from the commencement to the end of morbid manifestations. The tubercular cough is not the same from one time to another; the dyspepsia differs; the expectoration may be nothing at the commencement; the physical signs are not all found, nor with the same characters at the initial period as at the termination. The synthetic review of symptoms, such as has been the custom to expose classically, has no value for the clinician, who seeks only methods of being certain as to the diagnosis of each of these periods, so diverse, so opposite of tuberculosis. 3. Our only preoccupation is to determine the diagnostic and prognostic value of each symptom in the series of morbid evolutions of phthisis, without being burdened with a general encyclopædic description, which does not apply to any given case

at the moment of diagnostic uncertainties. Instead of making a synthesis from symptoms, we must analyze each in its period and place, which is the function of the clinician, and not to substitute himself for the pathologist.

§ 58. *Hæmoptysis.*

I. *Hæmoptysis in general. Frequency. Initial hæmoptysis.*—(a) Hæmoptysis is observed at the two extreme periods of the malady, at the beginning, and in the ulcero-cavernous period, more rarely in the intermediate period, so that one can say, in a general manner, that it is met with in two-thirds of the patients (Condie finds but twenty-four per hundred, Williams seventy per hundred). But what is required to be known is that, not only is it most frequent at the first period, but often constitutes the sole initial symptom. It is a warning that one observes often, in the midst of the most perfect health, more often again with individuals, who, according to their own opinion, have had for some time a slight and unimportant cough. Lastly, one sees patients who take their phthisis in an *hæmoptoic manner.*

(b) *Bloody expectoration.* — Sometimes we find but sanguinolent expectoration, mixed more or less completely with salivary, or mucous *crachats*, vitriform, or with certain streaks of blood, which result from the efforts of the cough. *Buccal sanguinolent expuition.* In one or other case the patients are seized with physical, or especially, moral anguish. They relate that, often on waking in the morning they find the mouth full of blood, or perhaps observe red stains on the pillow. Then, before sharing in the fears of the patient, inspect the buccal cavity, and you will often find the true cause of these sanguinolent expuitions, which must not be confounded with expectoration. The gums are softened—bleeding; they bleed at night by the efforts

at suction made involuntarily and unconsciously during sleep. In this case we have only to deal with sanguinolent saliva.

(c) *Hæmoptysis properly called. Its course.* — True hæmoptysis starts in general suddenly, and without cause, or follows an often insignificant effort. A cold bath, a forced march, a slight cold, a simple tickling of the larynx, and the patient loses fifty, a hundred, even up to a thousand grammes of bright blood, ordinarily frothy, and generally liquid. When the blood has stopped, the expectoration continues for several days to present a sanguinolent character. Afterwards a new loss of blood reappears once or several times, and all seems to happen in order. It has been advanced that an intermittent character has been found (Gerhard, Brehmer). The proof drawn from the favourable action of quinine establishes nothing. Patients are to be seen in which hæmoptysis has not reappeared, and nothing succeeded the loss of blood; others are seen remaining safe for a long time, setting at defiance, so to speak, the malady which menaces them.

(d) *Analogy with hæmatemesis.*—We suppose naturally hæmoptysis to be clearly recognized and separated from hæmatemesis. As at the end of violent paroxysms of coughing, especially with the subjects of tubercular disease, vomiting often occurs, and more especially when the blood comes in gushes into the mouth, the patients say they have *vomited blood*, whilst the case is simply that of hæmoptysis. To avoid this error we must immediately after the accident inquire into the nature of the expelled material. The blood effused into the bronchi remains in part retained in these passages (as proved by the bronchial râles), and does not pass out until the succeeding paroxysms, and this temporary sojourn gives to the blood a darker colour, reddish brown. All the blood that is eliminated

immediately is bright red. If, on the contrary, we have to deal with hæmatemesis, the blood is rejected altogether, presenting a black colouration, and a small part passes under the same form, that is to say, melœna by the stools. The blood of the stomach is often mixed with gastric juice, which gives it an acid reaction.

II. *Diverse origins of hæmoptysis.*—This problem once solved, we must learn the signification of hæmoptysis. Abstraction being made as to *infectious* maladies, *hæmophilia*, acute lesions of the lung (*pneumonia, abscess, gangrene*), aneurisms of the aorta, that is to say, of circumstances in which hæmoptysis is nothing more than an epiphenomenon more or less grave, confusing itself, so to speak, with the malady which has given it birth. We are, then, acquainted with only two real proven causes of hæmoptysis, to wit, heart diseases, and pulmonary tuberculosis. With women we must add a third possible, and even frequent, cause. This is disordered menstrual function, the hysteric state, with vaso-motor troubles.

(a) *Cardiac hæmoptysis.*—Hæmoptysis may be the first signal of cardiac maladies, especially mitral obstruction. It is enough to be warned of the existence of this phenomenon to recognize the origin of the hæmorrhage which is frequent enough, and is often accompanied by infarctus of blood vessels and emboli. It is also met with in distinct advanced cases of mitral or tricuspid lesions (see *Traité des maladies du cœur*, 1883).

(b) *Hæmoptysis supplementary or complementary to menstrual flow* is undeniable; it is found in women who, every month before, during, or after their period, present pulmonary hæmorrhage often sufficiently abundant, although rarely compromising life. If the coincidence is exact, and if in the interval of the periods, that is, of the hæmorrhage, the general health remains intact, without pulmonary

trouble, there is every reason to believe in functional hæmoptysis.

But in general we must distrust these derivations. Louis has observed hæmoptysis more frequently from forty to sixty-five years, that is, at the epoch of the menopause and after, than in the twenty preceding years; the number of supplementary cases of hæmoptysis is not such as to sensibly increase the proportion in women.

It is the same with hysterical subjects; hæmoptysis, *called nervous*, already recognized by the ancients and controlled by modern medicine, may have its point of departure in an authentic lesion of the nervous system (Ollivier, Bash, Labout, and Rosenbach), which may even be produced artificially (Brown-Séquard, Nothnagel), or rather in functional trouble of nervous system like hysteria. It is ordinarily easy to recognize, and is never of importance.

(c) *Arthritic hæmoptysis.*—Recently Huchard, recurring to the question of the origin of slight hæmoptysis, describes the hæmoptysis that occurs in gouty people, and that without relapse; but the proof of time is nothing, hæmoptoics are cured very often in a definite manner from first attacks of phthisis.

§ 59. *Mechanism of Initial Tubercular Hæmoptysis.*

People have spoken vaguely of bronchial or pulmonary hyperæmia; they have admitted a greater friability of vessels; lastly, they have supposed diapedesis. The congestion of vessels is not explained and explains nothing; the diminution in the consistence of vessels has never been seen; and as to the diapedesis it could never produce such enormous hæmorrhage as often to endanger life. The only explanation which remains is alteration of the vessels. Rindfleisch has demonstrated that in primitive granular tubercle one *constantly* finds that the arterioles and pulmon-

ary capillaries participate in the formation of the neoplasm. Perivascular tuberculosis, or rather tuberculous perivasculitis, which has been especially studied on the cerebral arteries, is shown by a neoformation of cells in the adventitious tunic, and by the fusiform tumefaction which is the consequence of it. These perivascular vegetations may accidentally penetrate the deeper coats of the artery and make them undergo tubercular degeneration. In a very clearly drawn figure Rindfleisch shows the invasion of a small pulmonary arteriole whose middle and internal tunic is perforated in a circumscribed point by tubercular perivascular infiltration. How, then, is the hæmorrhage produced? Let us recollect that the last branches of the pulmonary artery are *terminal arteries*, that is to say, beyond a certain point there is no more anastosmosis. If the channel of one of these vascular branches comes to be contracted, for example, by a perivascular tubercle which projects inwards, the general pressure of the vessel begins to increase, starting from the contracted point, and rupture takes place.

The opportunity is happily rare to study the mechanism of initial hæmoptysis, which is often stopped by the formation of a thrombus. Later, when the hæmoptysis indicates the presence of a cavity, it is quite otherwise; the hæmorrhage is often irresistible; the origin and danger comes from the cylindroid aneurisms which are found on the walls of cavities or from diffuse aneurisms of pulmonary artery which have formed in the cavity, and in which one finds cruoric clots rarely decolourized (Ramussen, Raynaud, Sevestre, Liouville, Damaschino).

§ 60. *Hæmoptoic Phthisis.*

I. *Long-standing hæmoptoic phthisis.*—When tubercles submit to fibrous transformation from the start, that is, whilst they are still in the granular state (Grancher) the meta-

morphosis is cure. On the other hand, the bacillus becomes localized more often than we think ; it surrounds itself with neoplasms which cannot infect the economy, so long as there is not a reiterated persistent multiplication of the parasites. If, then, we find the bacillus in these cases as we nearly always do, and which proves the parasitic and consequently tubercular nature of the hæmoptysis, that is not absolutely fatal, the bacillus may be arrested in its evolution. After pulmonary hæmorrhage the râles caused by blood filling certain bronchioles are not to be considered causes of alarm ; it is ordinarily absorbed after a few days and its presence does not hinder the fibrous transformation of the tubercles which have provoked extravasation of blood.

Hæmoptoic phthisis of rapid evolution.—When there is scarcely an interval between the attacks of hæmoptysis and progressive caseation and ulceration of tubercles, the cases are dangerous. Nothing will stop hæmoptysis from small cavities whose walls are covered with aneurisms, and of which the incessant rupture provokes the loss of blood.

§ 61. *Diagnosis of Hæmoptysis.*

Nervous hæmoptysis.—This denomination is only justified after the most minute and repeated examinations of the lungs, after the most severe inquiry into the state of nutrition of the patients. All bodily failure, all alteration of respiratory murmur is a cause for revision, or reform of a pronounced judgment. When women lose blood at their menstrual periods, at once by their lungs and from the uterus (complementary hæmoptysis), the principal danger is from the hæmorrhage causing anæmia. Tubercles are not so frequent as in—

Supplementary hæmoptysis.—Andral says this : " Nearly always when I have seen women spit blood at each menstrual period, I have assured myself they had tubercles. It is not

always a capricious deviation of the uterine loss; far from it. The menstrual flux has been suppressed by pulmonary tuberculosis; the lung becomes a seat of a double congestion that is due to the tubercles acting on the vessels of the lung in a manner to compromise their integrity; and on the other hand, what one calls the hæmorrhagic molimen, a sort of plethora resulting from the retention of the menstrual blood."

§ 62. *Latent Phthisis, with Dry Spasmodic Cough.*

The cough has three distinct forms; the common names are, dry cough, cough with vomiting, and catarrhal cough; the first finishing at the end of a certain time, often of very long duration, by being transformed into one of the other forms. The last depends on local or general bronchial catarrh, and will serve us for the occasion of its description. The physiological mechanism of coughing must be borne in mind, also tussigenic zones or regions. The laryngeal, tracheal, and bronchial mucous membranes are all excitable from mechanical irritation, also the pharynx and eustachian tube. The pleura is probably excitable, but not the pulmonary parenchyma itself or stomachal mucous membrane.

§ 63. *Paroxysmal Dry Cough.*

Ordinarily the cough of consumptives is dry; that is, without notable expectoration, without râles on auscultation and of a dry *timbre;* it is short, formed by a few sonorous expirations, whose union constitute interrupted jerks, which are repeated once in two minutes at most. In certain cases there is a considerable analogy with hysterical cough, or with the paroxysms of whooping-cough. Often the cough is most troublesome at night, especially during the first sleep; in the second half of the night it ceases.

Mechanism of short cough and of paroxysmal cough.—

Whether nocturnal or diurnal the cough always constitutes a reflex phenomenon produced by the bronchial secretion; ordinarily dry, scanty, adherent at the beginning, this exudation, perhaps already containing bacilli, produces on the mucous membrane, or rather on the sensible nerves of the bronchi (terminal fillets of the pneumo-gastric), an excitation which by centripetal path gains the bulb and is reflected by the expiratory motor nerves, amongst others by the motor nerves of the muscles of the abdomen. As clearly shown by physiology, the cough has no relation with the pulmonary lesion, it is set up by bronchial exudation, or possibly, in some cases, by adenitis of glands in the mediastinum which compress and irritate the vagus nerves in their passage. Cold air, or an atmosphere containing irritating particles, and perhaps also the entry of air into the respiratory tracts, its passage through the glottis and entry into the bronchi, will be the principal cause of the fits of coughing.

§ 64. *Paroxysmal Cough with Vomiting of a Mechanical Order.*

According to Morton, this is one of the most certain signs of initial phthisis.

Character of vomit.—Generally intact food or mucus and gastric juice only when the stomach is empty. The vomiting is the result of expiratory efforts, and has no connection with dyspepsia.

§ 65. *Diagnosis of Latent Phthisis.*

Its insidious beginning may give rise to errors: 1. As to a simple cold, and this causes grave negligence. 2. As to a nervous cough, which suggests extremely imprudent methods of treatment.

Cold.—Suppose that without known cause (if there is not

a legend of cold or damp) in the midst of perfect health and good hygienic conditions, an adolescent or adult of either sex is taken with a dry repeated, persistent cough, which resists all treatment, or with a painful paroxysmal cough, followed or not by vomiting and with or without bitonal dysphonia. As Jaccoud says, "How shall we distinguish the origin, the nature of this cough?" Only by repeated explorations of the lungs, to ascertain the absence of all modifications of respiratory murmur, and all vestige of localized râles at the apices. It is very rare that the immunity of the lungs should be complete for long, that one does not find a change, that is, a weakening of inspiration, or jerky respiration, or rude and prolonged expiration. If it is thus, if the respiratory trouble attacks an individual affected with general denutrition, suspect tuberculosis, and act accordingly.

Nervous cough.—This is absolutely continued and irresistible, without the intervals that one observes in the paroxysms of tubercular cough.

It *always* stops at night, contrary to what is seen with phthisical patients, but may last whole months without at all troubling the general health, and without producing the least expectoration or the least alteration of respiratory murmur. Its sound is *very sharp*, and with an elevated tonality which is invariably the same.

XXIII.

LATENT CATARRHAL PHTHISIS.

PHTHISIS is very often for a long time characterized only by a cough which, after having been dry, painful, and paroxysmal at the commencement, takes the catarrhal character. The patient expels certain rare *crachats*, which are composed of mucus differing in no way from that of

ordinary bronchitis, except that they are often marked by streaks of blood. When phthisis takes the catarrhal form immediately or after having passed through the painful and prolonged form of irritative cough, it presents the most complete analogy with bronchitis, whatever be the course, duration, succession, or repetition of such catarrhal inflammation.

§ 66. *Diagnosis of Latent Catarrhal Phthisis and Bronchitis.*

Phthisis may take the form of the most simple influenza, of tubercular or chronic bronchitis, and of repeated chronic bronchitis; in the latter case it is necessary to decide if tuberculosis is the cause of the evil, the reciprocal condition being impossible. Bronchitis never developes tuberculo-bacillosis.

Influenza is characterized by (*a*) an initial febrile state whose temperature curve is gradually ascending; (*b*) premonitory coryza; (*c*) disseminated râles, dry or humid; (*d*) an easy cough followed by a muco-purulent expectoration; (*e*) if the cough is prolonged, it may take the paroxysmal form, but even then it preserves the same characters. Tuberculosis, when it is febrile at the beginning, which is rare, never presents the regular tracing or the early decreasing course of influenza; the râles are rarely sibilant, and if they are generalized they are not long in concentrating themselves at the apices, unmasking thus the local irritation produced by the tubercles, whose chosen place is precisely the upper lobes. The cough, even if there are râles, is rarely followed by expectoration, and more rarely preceded by coryza (Louis, Walshe).

II. *Subacute or chronic bronchitis. Diagnosis by antecedents.*—When we have to deal with subacute or chronic bronchitis whilst it remains bilateral and characterized by

sibilance, it is impossible to recognize in it a tubercular origin. It has been well said that when it is manifested in a vigorous subject, without hereditary taint, we may in all security turn away from the suspicion of tuberculosis. It seems that to cause the least anxiety as to the future of a patient, his constitution must be deteriorated, his health be altered at the first attack, and that hectic fever has started at the first manifestation of bronchial irritation. It is clear that if one judges the patient by appearances, or the malady by signs already too clearly marked, then, in the latter case, the diagnosis is imposed; in the former it is impossible to expect that pretubercular bronchitis should attack an individual full of strength and with the best certificates of origin. The diagnosis, then, is only possible when bronchitis after generalization concentrates itself at the apices. The problem, then, is decided against the patient, whoever he may be.

Pretubercular bronchitis.—Laënnec says, with marvellous clearness, "This old opinion of a cold *badly treated* or neglected degenerating into pulmonary phthisis, is but a false application of the *post hoc, ergo propter hoc.*

"If the first symptoms are those of pulmonary catarrh, it is because tuberculosis exists in an apparently latent manner, but marked in reality by all the physical signs of tubercle; this catarrh is produced by the irritation that the tubercles exert on the lung as foreign bodies; no one has ever been able, scalpel in hand, to show the passage of catarrh, which is *bronchial*, into tubercle, which is *pulmonary.*" Most cases of phthisis, those which are most plainly tubercular, amongst others granular phthisis, are developed without the least bronchial accident; often even they pass through all their stages without provoking the least congestion or causing the least bronchial irritation. All these reasons are irrefutable, and I have but one rec-

tification for the opinion of Laënnec, which is, that if catarrh is produced by the irritation of foreign bodies, ought not one to admit also that it may be the product of the direct action of the bacillus, and that, consequently, bronchitis may precede tubercle as bronchitis, before being *tubercular bronchitis;* it may appear simple from an anatomical point of view, although in reality it may be specific as to causality; it is parasitic bronchitis, as there is bacillary laryngitis, an intestinal microphytic catarrh; tubercle is not long in following these toxic irritations. In this case it is not bronchitis which determines the formation of tubercle; it is the preface of more profound imprints that bacillosis leaves in the living organism.

Peritubercular bronchitis.—When tubercle invades the bronchi it inflames the mucous membrane in the cellular, epithelial, glandular, adventitious, and lymphatic layers, that is to say everywhere, especially in the bronchioles. Cylindrical epithelium, as Grancher observes, resists for a long time often when the cavity of the bronchus is already half full of epithelial cells and pus; the sub-epithelial tunic is swollen considerably, and transformed into a ribbon-like lamina. In chronic bronchitis this disappears, and the epithelium rests naked on the cellular tunic, which is itself the seat of an embryonic infiltration, granular vegetations, and ulcerations. The muscular tissue is invaded, in its turn, diminished, or even destroyed, so that the bronchial wall resists no longer, but dilates. This bronchial dilatation is scarcely found but in chronic fibrous phthisis. The bronchitis is generally limited to the neighbourhood of the tubercles, and is consequently easy to recognize.

§ 67A. *Latent Phthisis with Dyspnœa.*

This symptom depends on very diverse causes, which are very difficult to appreciate.

Character of dyspnœa.—Most frequently gradual, it is manifested especially under the influence of the cough, walking up-hill, repletion of stomach, to disappear with rest or repose in bed, particularly some hours after digestion. It often happens that it is clothed with quite another character; it is revealed suddenly by an attack, like asthma, morning or night, under the influence of variations in temperature or horizontal decubitus. Phthisis, then, may resemble asthma, not only by the course of its attacks of oppression, but also by physical signs, that is, by sibilant râles and tympanic sonority, which denote bronchial catarrh and emphysema. We shall see how to distinguish asthma from this tubercular pseudo-asthma. In children these violent attacks, with a whooping cough, are often the expression of engorgement of bronchial glands, and the compression they exercise on vagus nerve; but it is still an obscure point in diagnosis.

Causes.—These are partly diminution of respiratory area, and partly excitability or excitement of terminations of vagus nerve. The diminution of respiratory surface neither accelerates respiration nor makes it difficult, unless it is suddenly established in an appreciable extent. Thus if there comes subacute catarrh, it often takes the suffocating form, and the dyspnœa is in relation with the mucus accumulation in bronchi, as witness the production of râles, the diminution of respiratory murmur, as well as other signs revealing the existence of a physical obstacle to the entry of air into the pulmonary alveoli. This, then, is dyspnœa of mechanical order. Suppose, again, pulmonary congestion provoked by the presence of tubercles, whether bordering or not on hæmorrhage: it restrains the area of pulmonary circulation, and hinders the respiratory changes between the gases of the blood, and those of the atmosphere; it is still mechanical oppression. Tuberculosis by itself

only embarrasses respiration when it is suddenly established, and invades to a great extent the bronchioles and the alveoli. This may be the case with granulations, and the absence of being accustomed to their presence as well as nervous influence must be invoked; for, in reality, the granulations, however general their invasion, neither obstruct nor compress the bronchi, and do not compromise the circulation in the capillaries. When these granulations appear slowly, in a state of dissemination, although even the eruption becomes general, they leave respiration free; it is then an affair of custom, as in pleurisies, which slowly compress the lung without being betrayed by difficulty of respiration, whilst in pneumo-thorax, which consists of the sudden irruption of bronchial air into the pleural cavity, the brutal compression of the lung is shown by violent orthopnœa. There are, then, two types of granulations, and further, in the form which produces dyspnœa, there is the necessity for the intervention of the nervous system. In women and children and feverish patients the respiratory movements are rather only accelerated than effected with difficulty; they often lose by superficiality what they gain in number.

§ 67B. *Thoracic Pain.*

This is another phenomenon which by its variability and mobile localization does not permit any diagnostic conclusion.

Character of the pain. — Ordinarily the suffering is referred to the top of the chest, in front, below the clavicles, also the patients complain of intermittent or permanent pain in the dorsal scapular region, the attacks being often increased by pressure or percussion, or by the sensation of fatigue. They are exaggerated also by movement or coughing, and do not follow the anatomical course of the nerves.

Nature and cause of pain.—Most frequently the starting-point is in *pleural adhesions ;* the parenchyma of the lung counts for nothing ; the most intense inflammatory hyperæmia of the lung does not usually cause pain unless the pleura is affected. Adherent pleuritis, particularly at the apices of the chest, constitutes the rule. Pleurisy may, however, be established without provoking the least sensation of pain. This happens generally in the dry subclavicular pleurisies which occupy the points most reserved from inspiratory efforts, the most fixed during respiration, the best sheltered from the straining of the cough ; that is why one so often sees consumptives who do not complain of any pain. But if the pleural inflammation is early established, and especially if with rapidity, one has the right to suppose that it irritates or compresses the nerves of the vicinity ; this is the origin of most of the pain.

Myosalgia : muscular fatigue.—This is the dominant character of debilitations in general ; to it should be especially referred such pain as occupies the dorsal scapular region. The same sensations are to be found on the perimeter of the chest, and at the attachments of the diaphragm, particularly in patients who complain of difficulty of breathing, or a troublesome cough. It is then, especially, myosalgia by functional fatigue.

XXIV.

PHTHISIS DOUBTFUL ON AUSCULTATION.

§ 68. *Auscultation in general.*

(The complete account given in French Edition has been omitted.)

§ 69. *Pathological Modifications of the Respiratory Murmur.*

§ 70. *Different Physical Phenomena.*

By order of frequency in the first period, by chronological order, and consequently, therefore, by diagnostic value, we may categorize the signs drawn from inspiration. Prolonged and rude expiration appears but rarely, and then at a late period. It has never served to recognize the malady when the percussion sound and respiratory murmur were not in fault; it is preceded always by signs drawn from the inspiratory murmur. The alterations in the vesicular murmur of inspiration acquire a considerable value with the triple condition; (1) of being localized at one of the apices, especially the left, whether under the clavicles or in the supraspinous fossa; (2) of being fixed, that is to say, persisting in the same spot; (3) of not being modified by cough. If any one of these inspiratory modifications, whether weakening of the murmur, or its rudeness, or the jerky type, is to be found in an individual, presenting pseudo-chlorosis, or loss of flesh, or night-sweats, that is to say, an index of the general perturbation of the organism, the diagnosis acquires a real certainty which is well marked with the scrofulous patient, better still with the invalid who has a cough and thoracic or dorsal pains, and, lastly, most distinct of all with the patient who has had one or more attacks of hæmoptysis. Of these three kinds of inspiratory trouble, namely, the weakened murmur at the summit, rude and short inspiration, or, lastly, the jerky inspiration, whichever be first in date, it ought to be taken into account, and the decision of the clinician will depend on it. Up to this point there are but presumptive signs drawn from the general condition, or from respiratory functions undergoing alteration.

XXV.

DOUBTFUL PHTHISIS REVEALED BY THE PRESENCE OF BACILLI.

THE question which we raise, and which we stated definitely on December 4th, 1883, relates to all doubtful diagnoses, impossible in presence of traditional scientific methods. We describe a method which is not content with the modest task of completing an established diagnosis, that is nothing, but one that enables us to establish the diagnosis in an immediate manner, which is all the pre-occupation of the practitioner, all the future of the patient.

§ 71. *Latent Phthisis.*

(Many interesting cases are described by Germain Sée, showing that the specific bacillus is *always* to be found in cases of tuberculosis, and not in any other disease which may simulate it. The particulars have not been given in the translation.)

SECOND CATEGORY.

Distinct Phthisis.

Phthisis is only established by physical signs, that is, by auscultation and percussion, and is demonstrated by the presence of microphytes in the expectoration.

XXVI.

AUSCULTATION: BRONCHIAL RÂLES.

XXVII.

PERCUSSION IN GENERAL.

(In the French Edition these chapters contain much useful information, but which is to be found in the classical treatises on the subjects. This English work being intended for practitioners, they have not been translated.)

XXVIII.

ON THE EXPECTORATION OF DISTINCT PHTHISIS.

§ 93. *Diagnostic value of Bacillary Crachats.*

(a) *Exclusive signification.*—The matters expectorated by the phthisical patient, whatever be the period of his malady (often even at the start) will always, like tuberculous matter itself, contain bacilli, which constitute the most important element of tuberculosis. These materials, then, are unchallengeable witnesses of the malady, and constitute its characteristic. It is, so to speak, the signature of tuberculosis; and this is so true, that if you find it in the expectoration, or rather in any morbid product whatever, you may be certain by the presence, well and duly established, of bacilli, that you have to deal with a tubercular patient, and in the particular species with pulmonary phthisis.

(b) *Proof by the negative.*—Inverting the proposition, examine comparatively the expectoration coming from another disease. Then when you have acquired, by repeated examination, the proof of the absence of bacilli in the *crachats*, you may, without fear, conclude that you have not to deal with a tubercular lesion.

(c) *Proof of their virulence: inoculability and trans-*

missibility by respiration. Not only the parasite is there to witness the nature of the malady, and to multiply or consolidate our basis of diagnosis, but it further constitutes and is alone the true pathogenic cause of tubercle. It has an ætiological value, such that we can explain by it how inoculated *crachats* are capable of reproducing all the series of tubercular alterations, and how, on the other hand, the inhalation of expectorated tubercular products is so fatal to men and to animals which are obliged to breathe this bacilliferous atmosphere. There is the poison all entire; it is the virus in a state of isolation, which acts in the liquid or solid substances coming from the phthisical patient. One comprehends after that how important it is to recognize the bacillus in the *crachats* at different periods of the malady.

(d) *Examination of bacilliferous crachats.*—In all our researches we have employed the method of Ehrlich, which applies to the study of the *crachats* as well as to that of tubercle itself. We place the *crachat, flattened out, and dried* between two glasses, in a capsule previously passed through the fire. Afterwards, one pours into it the colouring solution, fuchsine. In half an hour the preparation is decolourized with dilute nitric acid; afterwards it is washed again, then coloured for half a minute with an aqueous solution of methyl blue, and mounted in Canada balsam. In this way the cells of micrococci are coloured blue, the bacilli are of a deep red; the most beautiful forms are furnished by the compact lenticular bodies in certain expectorations; it is there one sees the bacilli in greatest number.

§ 94. *Search for Bacilli in Distinct Phthisis.*

The most considerable work on this subject is that of *Balmer and Fräntzel,* whose conclusions are as follows:—1. In 120 cases of phthisis, bacilli were found 120 times, whilst they were totally wanting in other patients attacked with

pulmonary affections. 2. Ordinarily, *crachats* rich in bacilli indicate a grave case. 3. The fever is in some respects a sign of the number of bacilli; but it must be clearly understood that the diagnostic signification of bacilli is of much more importance than their prognostic value. Researches of this kind, however, have been undertaken in every country of the world, especially for clinical purposes. Contradictory results have been very seldom arrived at.

§ 95. *The Expectoration in Different Periods of Tuberculosis.*

Composition of crachats.—The observations of Renk state that the mean quantity of expectoration daily was 124 grammes.

Average Composition.

Water	94	
Solid parts, organic ...	5	2 Mucin. 2. Extractive matters. 1 Albumen and Fat.
Inorganic	1	NaCl, phosphates, etc.

Histological composition. As ordinary structural elements, we find globules of pus, red blood corpuscles, pavement epithelium, little drops of myeline, very often crystals of cholesterin. Professional phthisis is marked also by the presence of different metallic or vegetable dusts in the *crachats.*

Special morphological elements.—These comprise alveolar epithelium and elastic fibres. The former acquires significance when in great quantity, and when coinciding with catarrh of superior lobes. In general this alveolar epithelium is in the form of rounded or more or less angular cells, subject to fatty or myeloid degeneration, and which may be partially decomposed, so as to set at liberty fatty granula-

tions or drops of myeline. Sometimes they constitute nearly the whole of the structural elements of the *crachats*.

Parasites.—The expectoration nearly always contains parasites; one finds there sarcinœ, the fungi of moulds, and often schizomycetous fungi which are met with in the granular detritus, and, according to Buhl, contribute singularly to favour the caseous transformation. Actinia have been described which constitute a special form of parasitic pneumonia, called pneumomycosis.

Materials from destruction of lung. Elastic fibres.—One of the most constant characters of pulmonary lesions, whether tubercular or gangrenous, consists in the presence of elastic fibres coming from the lung, which is in process of destruction, often even before physical signs have revealed anything of the morbid process. In advanced periods it denotes the progress of the malady.

Origin of crachats.—These are generally abundant, and consist in great part of muco-purulent material, which is not always easy to distinguish from bronchial expectoration. In reality, a part of the excretion comes from the bronchial mucous membrane attacked with concomitant catarrhal inflammation. Another part is formed by the purulent secretion of the walls of the cavity, and in these cases the expectoration has a tendency to agglomerate in rounded masses called *nummular crachats*. Often the purulent exudation is intimately mixed with blood, which is caused by the mixture of pus with blood coming from the rupture of little capillary vessels, and this mixture takes a dirty reddish brown or chocolate colour.

THIRD CATEGORY.

Masked Phthisis.

Under the name of masked phthisis we shall describe: 1. All those which resemble common diseases of respiratory organs—that is, of bronchi, lungs, larynx, pleuræ, and the organs of circulation; 2. All cases of masked phthisis which are announced by bacillosis of the digestive organs or genito-urinary system.

XXIX.

MASKED PULMONARY PHTHISIS.

Phthisis may start as an inflammatory or congestive lesion of the lung, that is, pneumonia, bronchitis, or congestion. Here are two observations on cases of pneumonia, apparently legitimate, but in reality bacillary.

I. *Acute phthisis under the form of simple lobal pneumonia.*—Observation No. 10, 1883. At number 17 in the ward St. Christopher, we treated a printer, aged 19 years, who on Sept. 13th was taken with a pain in the side, shivering, fever, and on the morrow he presented himself at the Hôtel Dieu, with all the signs of acute lobal pneumonia, occupying the superior two-thirds of the right lung. At the end of nine days, as defervescence was not clearly established, we examined the *crachats*. They contained a considerable quantity of bacilli. In the first few days of October we found cavernous gurgling, amphoric *souffle*, in a word, the complete semeiology of phthisis, which had started by lobal

pneumonia, and in three or four weeks had passed through all the stages of extremely acute phthisis.

Observation No. 11. Here is another case (St. Christopher, No. 4), much more complicated and interesting; I add, more decisive of the diagnostic value of bacilli. It is that of a man who had pleurisy on the left side, and who came into the hospital for pneumonia (right), which was considered simple and legitimate. At the end of three weeks the pneumonia not arriving at complete defervescence, we began to be afraid of having to deal with tubercular pneumonia, and sought for the bacilli. A first examination showed only the presence of a large number of common bacteria; it was not until the third examination that we were able to show the presence of the bacillus tuberculosis. He was then a pleuritic who had become tuberculo-pneumonic.

I find in the publication of M. Cochez an analogous observation with reference to a woman attacked with right lobal pneumonia.

II. *Phthisis in the form of acute bronchitis or influenza.* —This is much more common and difficult to recognize. We have recorded several examples.

III. *Phthisis in congestive form.*—Relations between phthisis and pulmonary congestion.

§ 96. *Diagnosis between Tuberculosis and Simple Bronchitis by Local Signs.*

Simple bronchitis is never exactly limited and in a fixed manner to the apices of the lungs, and especially to *one*, in the same way that tubercular bronchitis is never equally or indifferently spread over all parts of the lung. Where tubercle is, there peribronchitis, or bronchitis, dominates; naturally it is from there that catarrhal irritation may gain the entire surface of the bronchi. Percussion may also facilitate the distinction between the two kinds of bronchitis. With

tubercle, even when primitive, the apex presents a very clear tympanic sound, on account of the expansion of the pulmonary tissue which surrounds the tubercles. This increased resonance may contrast with the normal sub-tympanic sound on the healthy side of the chest. The third distinctive sign is the absence of emphysema in tuberculosis and its nearly inevitable presence in chronic bronchitis. In reality, emphysema is never developed except in advanced periods of chronic phthisis when there can be no difficulty of diagnosis.

§ 97. *Pulmonary Congestion, its Relations with Phthisis.*

(The truth is simply we have to deal with secondary congestion, which attacks the pulmonary parenchyma already tuberculized.)

§ 98. *Diagnosis of Masked Phthisis of Pneumonic Form. The Relations of Tuberculosis with Pretubercular and Tubercular Pneumonia.*

I. *Diagnosis of tuberculosis and acute or chronic pneumonia.*—(*a*) *Acute pneumonia.* When the practitioner is called on to decide between tuberculosis and declining acute pneumonia of the apex, hesitation should not long exist; even supposing that one is ignorant of the recent and sudden start of the malady, its extremely acute course, the initial intensity of the fever, with the pain in the side, and viscous, bloody expectoration, one is always able to establish, by manifest slight dulness at the apex of the lung, the tubal murmur, and especially by the fine crepitant *râles* at the beginning and afterwards by the redux *râles*, that we have to deal with a transitory accidental malady which has nothing in common with tuberculosis.

(*b*) *Pneumonia with imperfect resolution.* If the fever has disappeared for two, three, or four weeks, we ought to refer

exclusively to physical signs and the diagnosis is still possible, for we never find in such a short time any considerable condensation of pulmonary parenchyma from tubercular infiltration; and if it should be so, by the caseous softening of this tubercular mass, it would be immediately manifest with sub-clavicular foci of dry cavernulous *râles*, which would be easy to distinguish from the redux *râles*, and, for a stronger reason, from the crepitant *râles* of pneumonia.

(*c*) *Chronic pneumonia.*—Latterly chronic pneumonia and cirrhosis of the lung have been described and are recognized by the absence of the bacillus (Debove, Ollivier, Ducastel). Acute pneumonia never terminates in a chronic form.

II. *Tubercular pneumonia.*—We have seen that pneumonia is legitimate or tubercular; the so-called pretubercular pneumonia does not exist. After Wunderlich and Sydney Ringer, all authors admit that by the sole fact of their presence in the lungs and still more so at the period of caseous degeneration, tubercles constantly determine an elevation of temperature. We have curves of vesperal fever called hectic which have no relation to those of pneumonia, especially of the lobal form. Fever is not synonymous with inflammation. The tubercular pneumonic forms are two.

Catarrhal pneumonia. Caseous pneumonia. Characterized as ordinary catarrhal pneumonia, that is, by congestion of vascular network, by the swelling and afterwards by the fall of the epithelial cells which have become granular and fatty, by leucocytes and red globules, catarrhal pneumonia is ordinarily disposed exactly in a zone round the tubercle. Once developed, it may attack several contiguous lobules and affect the pseudo-lobal form, containing everywhere, however, the granulations. According to Cornil and Rauvier, it occupies the same regions as simple broncho-pneumonia—the inferior part of upper lobe, the posterior border and base of

inferior lobe. The difference is less, as one does not there find always the evidence of the primitive malady, that is, the granulations. Whatever it be, it generally passes into the caseous form, or rather at the peripheric part of the catarrhal inflammation, one sees the embryonic zone, the characteristic precursor of tuberculosis. Starting from this moment, it enters into the domain of tubercular infiltration; having become caseiform, there does not remain the least trace of divergence. As Charcot says, supporting his assertion logically from facts, in the majority of cases the great destructive process of the lungs is independent of pneumonia, and the principal symptoms of phthisis have no connection with the inflammatory element which may be wanting during all the evolution of the malady. The identity of catarrhal pneumonia which has become caseous, with massive tubercular infiltration may be considered as demonstrated.

XXX.

MASKED PHTHISIS RECOGNIZED BY BACILLI. PSEUDO-PHTHISIS RECOGNIZED BY THE ABSENCE OF BACILLI.

(After giving several observations, both negative and positive, as regards bacilli, M. Sée goes on to add that, in presence of pseudo-phthisis, that is pleurisy, nephritis, spasmodic cough, hæmoptysis, which have given good reason to believe in the existence of tuberculosis, the absence of bacilli will enable us to give a positive opinion against tubercle. On the other hand, we see in his last three observations phthisis simulating typhoid fever, metro-peritonitis, simple pleurisy with friction. Nothing would have made us suspect the tuberculous nature in such complex cases. The presence of bacilli alone permits the affirmation.)

XXXI.

MASKED EXTRA-PULMONARY PHTHISIS.

§ 99. *Affections of the Larynx. Relations with Tuberculosis.*

THE larynx in tubercular patients may be affected in different fashions.

1. It may be the seat of catarrhal tumefaction, which profoundly alters the voice.

2. There may be *anæmia* of mucous membrane, and especially of the glottis, and this is still but little studied.

3. The compression of the recurrent nerve by one of the swollen bronchial glands is one of the most frequent causes of aphonia. In this case the paralysis of posterior arytenoid muscle leaves the respiratory glottis open during phonation. Paralysis of the internal thyro-arytenoid determines a gaping condition of ligamentous glottis after the emission of the voice. The nerve itself may be compressed by cicatricial adhesions of the pleura with or without pulmonary sclerosis.

None of these alterations are special to tuberculosis, and should not be put down to the agency of laryngeal phthisis.

4. The true ulcero-tubercular laryngitis.

All these laryngeal affections, especially the properly called laryngeal phthisis, constitute a grave and frequent complication of tuberculosis.

Correlation with pulmonary phthisis.—But there is a preliminary question to decide. Is the ulcerative process a primitive manifestation of tuberculosis, or is it only a

secondary result of pulmonary tuberculosis, imperfectly indicated or misunderstood? The first opinion is not doubtful. The malady may proceed from the larynx to the lungs. Sommerbrod has experimentally demonstrated the progressive invasion of the lungs following simple or ulcerous irritation of the larynx. One may even say that it is not rare to see pulmonary tuberculosis announced by simple or tubercular laryngitis, which remains isolated during a certain time. It may also be that, when tuberculosis attacks manifestly both organs, that the laryngeal alteration dominates, and puts the pulmonary lesions in the second place.

Primitive lesions.—Primordial laryngeal phthisis has been long denied; but observation shows that individuals, in appearance perfectly sound, begin by presenting a modification of the voice, a more or less complete hoarseness, which is at first treated as common laryngitis, but which resists all treatment. Other troubles are manifested in the larynx. The general condition gets worse. The laryngoscope shows at first only congestion, simple catarrh of the vocal mucous membrane. It is not until much later that we discover the ulcerations and infiltration of the mucous membrane. During all this phase, which may be long, the most minute examination of the chest furnishes no proof of the existence of pulmonary lesions. This immunity is not indefinite, and it has been asked if the laryngeal affection has followed pulmonary tuberculosis, which had escaped our means of investigation. But nothing proves that the tubercular poison may not bear primitively on the larynx, determine there the first microphytic manifestations, and not attack the respiratory organ until a later period. It is true that in most cases the order is inverted. The vocal organ is not attacked until after the development of pulmonary tubercle. In this case

may we not admit that the tubercular *crachats*, in passing over the mucous membrane, produce the laryngeal alteration?

Enumeration of lesions in the larynx. Erosions. Tubercular infiltration. Tubercular ulceration. — The latter is found principally on the arytenoid cartilages, vocal cords, and on the epiglottis. We shall study successively laryngeal catarrh, paralysis of vocal cords, and ulcero-tuberculous phthisis.

Acute and chronic catarrhal laryngitis.—The signs and symptoms are as follows:—

1. The sensations of tickling and excitation in the larynx, or of tearing or roughness, which lead the patients to cough.

2. The cough is generally hard and without expectoration. It is not till later that the patients eliminate a transparent, vitreous, watery mucus, containing, when seen under the microscope, certain muco-purulent corpuscles mixed with epithelial cells, rarely with ciliated epithelium. On the addition of acetic acid the globules of pus swell and become transparent, which enables us to recognize from one to three nuclei. At the same time the mucine is precipitated as a flaky cloud. Later this primitive expectoration is transformed, diminished in mucine, increased in purulent corpuscles, and augmented in quantity. Sometimes it is mixed with streaks of blood, especially if the cough is violent and the inflammation very pronounced.

3. The cough at starting is generally rude, sometimes spasmodic. The spasm, after a strident inspiration, seems to be due to sudden complete occlusion of the glottis, and to be expressed by shocks, which succeed in forming a veritable series. Is not this also the character of pre-tubercular cough in general?

Voice.—The voice is constantly altered, and the change may take place in four different ways.

(*a*) Swelling and inequality in vocal cords.

(*b*) Mucus accumulated on free borders of glottis.

(*c*) Swelling of false vocal cords.

(*d*) Nervous paralysis, motor or sensory.

(The excellent description of the nervous physiology of larynx by Eichhorst is given textually in the French edition.)

Ulcero-tubercular phthisis of larynx. — The lesions in this affection may be divided into three groups: (1) Tubercles and tubercular ulcerations; (2) follicular and sub-epithelial ulcerations; (3) superficial erosions. It was Virchow who first made tubercle of the larynx understood.

The signs of ulceration.—In an extent which is often not greater in volume than the head of a pin, but which may comprise a great part of the mucous membrane, the ulcers are to be found, often rounded, more often irregular, sometimes superficial and only attacking the epithelial layer, but most often deep enough to present a funnel shape, or that of a crater, with tortuous borders and papillary excrescences on these borders of a yellowish or lardaceous aspect, rarely clean and red. These ulcerations occupy by preference the posterior wall in the inter-arytenoid region, often also the mucous membrane of vocal cords near their posterior attachments (more rarely the false vocal cords), the mucous membrane covering arytenoid cartilages, and frequently enough the epiglottis, which may be destroyed. With the progress of ulceration one sees the infiltration of neighbouring tissues developed, perichondritis, necrosis of cartilages, separation of attachments of vocal muscles, and lastly, what is most grave, œdema of the glottis.

Symptoms of ulceration.—After a laryngoscopic examination, which reveals all the peculiarities of the ulcer,

at least its extreme minuteness, or an ulcer on the posterior wall, or even the presence of an exudation which covers momentarily the ulcerated surface, then all the other signs have no pathognomic value, but contribute singularly to an exact diagnosis, and also often to an aggravation of prognosis. The voice is altered even to extinction, and if the examination by laryngeal mirror gives no reason for this aphonia, but reveals only an extensive or profound ulcer, it is that the aphonia is paralytic, or rather it is the last period of the malady; as Krishaber and Peter say, "it is an exhausted voice." The patients often complain of a painful sensation of tickling which provokes or precedes the cough, and more often sharp pain which extends to the ears, and is singularly increased by deglutition. A violent cough torments them day and night, and they finish by expectorating muco-pus (sometimes a trace of blood), which contains, under the microscope, elastic fibres differing from those of the lung, in that they are finer, and more rectilinear. The fibres of elastic tissue from lung are always flexuous and more convoluted. The most dangerous and painful symptom is dysphagia, resulting from the destruction of the epiglottis; the introduction of solid food, more often liquids, provokes infallibly at the same time paroxysms of cough; so that the patient ends by refusing nourishment, and inanition becomes imminent. Another danger waits—asphyxia. Œdema of the glottis puts an end to all these cruel sufferings.

§ 100. *Laryngitis and the Microphytic Examination of Mucus.*

(Fräntzel has described, in ten cases of laryngitis, the decisive importance of tubercular bacilli in *crachats*, which have been expectorated or taken from the larynx with the aid of a brush. Germain Sée gives the history of a case first diagnosed as tubercular in this manner.)

§ 101. *Pleural Phthisis.*

We observe before, during, and often at the decline of phthisis, different forms of pleurisy. It plays an important part, in an ætiological point of view, even as a premonitory lesion, and lastly as a grave complication. In all these conditions it may render diagnosis difficult; and that is what is always the case, whatever be the lesion which characterizes pleural inflammation. These lesions take all possible forms. There exists (1) a dry pleurisy, which is often initial; (2) pleurisy with effusion, which is sero-fibrinous, purulent, hæmorrhagic; (3) pleurisy in one or the other case may be tubercular; (4) lastly, special mention is reserved for the pleural adhesions which follow, and remain as vestiges of cure in dry or sero-fibrinous pleurisy.

I. *Dry or plastic pleurisy.*—It is not rare to find in the state of health, and for stronger reasons in the tuberculous, traces of dry pleurisy under the form of pseudo-membranes more or less extended, but without fixed adhesions, and having their seat on the costal or parietal pleura.

Generalized dry pleurisy.—Most ordinarily it occupies a very considerable space on the sides of the chest wall, and is expressed by two signs, namely, a dull sound, and the pleural friction-sound, which may be easily confounded with sub-crepitant *râles* and the humid cracklings of tuberculosis. But these friction-sounds, by the fact that they occupy the sides, and often the bases of the chest, should never be confounded with sub-crepitant *râles*, and especially with cracklings which are only found in the sub-clavicular or supra-spinous regions. And it is at most that they might be taken for the *râles* of bronchitis; for even when the humid friction-sounds recall by their pitch *râles* of such nature, they cannot lead us into error by this fact alone, that they

do not answer exactly to the two phases of respiration, and in no way yield to the efforts of the cough, whilst the *râles* are mobile, and generally more marked during one or other periods of respiration. The diminution of sonority, if well demonstrated, will be proof most in favour of pleurisy with soft and unequal plastic membranes.

Dry pleurisy of apex.—It is in plastic pleurisy, at the top of the chest, where one establishes best this obscurity of percussion sound, by direct reason of the thickness of membranous layers, which, so to speak, coif the lung. But we can comprehend the possibility of error, since disseminated tubercles do not alter the sound, and, on the contrary, give rise to a certain modification of respiratory murmur which the most dense, the most extended pleuritic layers, never trouble. It is with these dry pleurisies, after all, that we find real causes of error, because we generally attach to them the most sombre prognostics. They are considered as the prelude and the future seat of tubercle; but I know invalids who for long years have presented manifest signs of dry pleurisy, but never the least indication of tubercle. Thus pleuritic patches at the apex are not in themselves of prognostic significance. Ten years ago I had occasion to examine, with two distinguished colleagues, a young collegian who showed slight dulness at apex of the left lung; he was considered to be tubercular, but he enjoys to-day most perfect health.

II. *Pleurisy with effusion.*—Pleurisy with effusion is sometimes the premonitory index of tubercle, sometimes the effect of the malady. It often happens that pleurisy with effusion, more often even than dry pleurisy, is the revealing indication of tubercle. The pleuritic patients often become phthisical, but it is well to add that rarely phthisis succeeds effusion without interruption; there happen sometimes long years between this grave prelude and the drama. How

to explain this lull, if not by supposing the granulations of the pleura curable, and transformed into tissue without nodules, or by admitting that tubercle, which comes later, fixes itself by preference on that part of the respiratory apparatus which has already been damaged, and which is *motionless?* It is there that the bacillus fixes itself most easily. However it be, we must be distrustful of pleurisy, even of the form called rheumatic. Pretubercular pleurisy may, nevertheless, be cured, and really as quickly as pleurisy *a frigore.* The same reflection applies to the effusion which is produced even in the case of tuberculosis itself. It may be simply sero-fibrinous, and capable of being rapidly cured in a spontaneous manner, although that may be after tapping; but it is not the less true that properly called tubercular pleurisy is often hæmorrhagic, and most often purulent.

Diagnosis between pleurisy and cavities.—All these effusions, especially the sero-fibrinous, may simulate cavernous phthisis; and this is the manner. Thirty years ago, Chomel, afterwards Rilliet, Behier, and other observers, described *simple* pleurisies, which in their course, and especially at the decline of the malady, presented signs of cavities, that is to say, amphoric *râles*, cavernous voice, and sometimes even the metallic sound with or without the *bruit de pot fêlé.* As these phenomena are often found added on to those of pleurisy itself, we find the signs of pleuritic effusion, particularly in absolute dulness with *absence of vibrations* in a great extent of half the chest, or in its lower part; one succeeds easily in eliminating the idea of a vast excavation which has invaded a whole lung in its lower part.

III. *Tuberculous pleurisy.* — The pleura participates generally in tuberculosis, which proceeds from the lung to the pleura. In these disseminated or agglomerated miliary tuberculoses which are found in the pleura, it is generally

the seat of plastic exudation—the inverse may be produced; the granulations may invade the false membranes of the pleura in a primordial manner; in the two cases the physical signs relate to dry pleurisy without one being able to recognize the mode of invasion of granulations, that is the method of penetration of bacilli into one or the other tissue.

IV. *Adhesive pleurisy.*—In a great number of cases adherences are formed, whether at the level of the affected spot, or in all the extent of the pleura on the damaged side, or even on the two sides. It is difficult to recognize these adhesions if it is not when the lung has undergone fibrinous or sclerotic transformation; in these cases we find often depression of upper ribs, local deformity of the chest; the thoracic framework, the pleura, the lung, all are engaged in this general retraction of the tissues in the fibro-sclerous process which diminishes and atrophies the lung.

§ 102. *Diagnosis of Pleural Phthisis by Bacilli.*

Pleural phthisis slowly developed.—It may be that phthisis commences in the form of pleurisy; this often precedes the apparition of the first signs of tuberculosis during a time so considerable that it is asked if there is any true relation between the inflammation of the pleura and the tardy manifestation of tubercles. One sees phthisical patients who count a pleurisy in their antecedents going back for ten, fifteen, or twenty years, as others count an hæmoptysis; it is asked in like manner if pleurisy can be, with this long period of development, the *cause* or the first *manifestation* of tuberculosis.

(*a*) The first hypothesis does not bear serious examination; it is not explained how plastic or serous, or even purulent inflammation (unless itself tubercular, and from being propagated to the lung) has been able to determine a

store of purulent material in the lung, and to retain this depôt *secretly*.

(*b*) The second hypothesis is much more plausible; we comprehend easily that an individual, especially of tubercular race, should be attacked on two very distant occasions by tubercular infection: a first time the pleura; the second time the lung.

Objections.—People will doubtless object that tubercular inflammation is never cured; but we know now that tubercle, wherever it is found, in the bones, in the glands, in the testicle, in serous membranes as well as in the lung, may be transformed into fibrous tubercle, and this tuberculosis, localized elsewhere, having become sclerous, gives no more sign of its presence during the existence of the patient. Must we recall on this occasion the hæmoptyses which are so frequent in early life, and which, all coming from tubercles more or less distinct, finish by being definitely cured? Although here, again, the hæmoptoic, like the pleuritic, ought never to forget that he has been ill; the cure takes place the first time, but a relapse may occur after a respite of long years, and then pulmonary phthisis bursts into the midst of health which appears unassailable, the primitive incident having left no traces.

Immediate pleural phthisis.—It may happen, otherwise, that between pleurisy and pulmonary phthisis there is no interval, and that one succeeds the other in an immediate manner. In every way it is important that the practitioner should be decided about the nature and origin of pleural effusion, to establish the diagnosis, and consequently the data for prognosis. As often the pleuritic patient neither coughs nor spits, we are obliged to complete our researches and examine the liquid obtained from the pleura. An account given by Professor Vulpian is quoted, showing two important points. The first is relative to the difficulty of

finding the parasite in the effusion, whilst we can so easily establish the existence of bacilli in the expectoration.

The second remark bears upon the obscurity of physical pulmonary signs in pleurisy and their tardy appearance; the microscopic examination of the expectoration may then be of great service.

XXXII.

MASKED GASTRO-INTESTINAL, PERITONEAL, OR GENITO-URINARY PHTHISIS.

PHTHISIS may take the appearance of affections of digestive organs and their annexes; more rarely it starts by the genito-urinary organs.

§ 103. *Bucco-pharyngeal and Stomachal Lesions.*

Bucco-pharyngeal lesions.—Tuberculosis may be manifested by alterations of the mouth and throat (thesis by Barth). Numerous facts cited by Guttman, Fräntzel, are available to witness the presence of microphytes in these neoplasms; it is often the distant forerunner of pulmonary phthisis.

Stomach.—The stomach is often the primordial seat of tubercular digestive troubles; we find there later simple or tubercular ulcerations.

I. *Bacillary dyspepsia.*—We already know the perversions of stomachal functions, constituting the insidious beginning of the malady, without speaking of the vomiting which constitutes a simple motor trouble in the majority of cases.

Nature of the dyspepsia.—Must we attribute this chemical dyspepsia to a stomach catarrh, such as has been described in Germany, or to a gastritis with nipple-like projections of the mucous membrane, such as has been indicated by French

authors? Nothing is less certain; what is most probable is that the irritation is due to the presence of bacilli. When the dyspepsia is tardy, we may suppose that the bacilliferous *crachats* are swallowed by the patient; when it is initial, we are right to ask if the bacilli have invaded the stomach with the food. We may be certain in one or the other case that the microphyte cannot fructify in the gastric juice whose acidity is absolutely prejudicial to it, whilst if it passes into the intestine it may there provoke tuberculisation; fœcal matters may actually contain specific parasites.

II. *Tubercular ulcerations.*—The best proof of the difficulty that the bacillus finds in living in the stomach is the rarity of ulcerations and tubercular lesions of the coats of the stomach; when they are found there it is in general tuberculosis. The granulation is never there in a crude state; ulceration with indurated borders is the sole witness of tuberculosis; it exists near the pylorus, and is often accompanied by caseous engorgement of neighbouring glands. These ulcerations have been seen giving rise to hæmorrhage and even to mortal perforations. Amongst numerous authors who testify as to the perforations the observations of Duguet are conclusive, whilst those of Litten are remarkable by the complete absence of all appearance of ulceration in the rest of the digestive tube.

III. *Signs of tubercular ulceration.*—Hæmorrhage, the only certain sign of ulceration, does not indicate if we have to deal with ulceration of a tubercular or simple character.

IV. *The round and tubercular ulcers often coincide.*—After the researches and statistics of Dietrich, Steiner, Volkmann, we may admit that in a hundred cases of round ulcer twenty-two are accompanied by tuberculosis. Is this an accidental coincidence, being found on account of the extreme frequency of phthisis, or must we consider the ulcer as a cause of phthisis on account of the inanition it provokes? The inverse appears to be true (see § 149).

In reality, by troubling the pulmonary circulation and also that of the stomach, tuberculosis may produce the round or peptic ulcer, whose starting-point is emboli, or obliteration of small vessels. In these conditions the mucous membrane is attacked by the gastric juice, and is destroyed by auto-digestion.

§ 104. *Masked Intestinal Phthisis.*

In all mucous membranes (stomach, larynx, intestine) we may and ought to count in the number of pretubercular affections, not only tubercles and intestinal ulcerations of tubercular origin, but different morbid states which at first sight appear strangers to phthisis.

I. *Premonitory or early diarrhœa.*—In reality we see patients at the commencement of phthisis presenting only that simple diarrhœa of chronic form, which is called intestinal catarrh in Germany, enteritis in France, while in reality it must often be referred to a simple motor trouble of intestine, like the vomiting without gastritis at the beginning, which really constitutes a dynamic effect, and is an important sign of tuberculosis.

How is diarrhœa produced?—Have we to deal with a primitive local action of bacilli introduced into the digestive tract with the aid of vitiated food; for example, the milk of tubercular cows? This question of origin has not been definitely answered, but the fact of initial diarrhœa is unquestionable. Do we not see these exaggerated secretions as modified by superficial irritation of mucous membrane? Is not the diarrhœa, like flux by excess of contractility, or contractions of intestine in the first few days of infectious fevers—typhoid, measles, malaria—such that in consequence one might suspect any ulceration whatever? The analogy is striking; in children, especially, pre-existing diarrhœa masks tuberculosis at its origin. In the adult, when diar-

rhœa persists with fever and loss of flesh, we must, says Chomel, be afraid of phthisis; thus it is found that even when the diarrhœa is prolonged, profuse, continued, and painful, that we see but insignificant lesions at the autopsy (Spillmann). There is, then, simple diarrhœa and spasmodic diarrhœa.

II. *Diarrhœa by simple follicular or peptic ulcerations.*—Another kind of intestinal lesion, which has neither the intestinal tubercle for origin, or evidence, is the state of inflammation of lymphatic follicles, which commences by tumefaction or inflammatory œdema, continued by inflammatory hyperplasia of cellular elements, and often terminated by superficial erosions. The mucous membrane itself may become the seat of ulcerations which are called catarrhal, and which generally invade the end of the small intestine and the colon, whilst follicular ulcerations habitually occupy the colon.

Peptic ulcers.—There is another kind of ulcer, which, perfectly analogous to the round ulcer of the stomach, is frequently produced in the duodenum; I call it peptic ulcer, because, following circulatory troubles of the mucous tissue, especially intestinal emboli, the digestive juices of the duodenum attack and destroy the mucous membrane; it is an auto-digestion. Attention has not been sufficiently directed to the correlation of tuberculosis with ulcers.

III. *Coliquative diarrhœa by amyloid degeneration of vessels.*—The ulcerations of which I have just traced the origin and nature are generally the precursors of pulmonary tuberculosis; those which remain for me to describe mark the end of the malady; these are the lesions and ulcerations coming from amyloid degeneration of intestinal vessels. The transformations which in the last period attack the liver, kidneys, spleen, muscles, may reach all the vessels, and those of the intestine are not spared. There is, then,

incurable diarrhœa from the formation of ulcers, which are caused in the same way as peptic ulcers, that is, from trouble of circulation by partial anæmia resulting from the obliteration of vessels, or by arrest of circulation; all these diarrhœas, which exhaust the patient so as to cause death, are called colliquative.

Résumé.—In enumerating the diarrhœas and ulcerations of a non-tubercular character in phthisical patients, I have endeavoured to demonstrate once more that *all is not tubercular in tuberculosis;* this warning is the more important, as discouragement is often spared the patient at the first appearance of diarrhœas. With the exception of diarrhœa of degenerative origin, called colliquative, all may be modified by well-directed treatment, which is based on the fundamental doctrine that in tubercular patients there are simple catarrhal, or even vaso-motor diarrhœas, to be combated by the common methods that all intestinal diarrhœas require.

IV. *Ulcero-tubercular diarrhœa of intestine.*—After this long preamble, I come to intestinal phthisis, that is to say, tuberculosis, and the ulcerations which result from it.

(a) *Relations with pulmonary tuberculosis.*—Tuberculosis of intestine, in a great number of cases, is a secondary malady, and constitutes one of the manifestations of general tuberculosis. Pulmonary tuberculosis precedes it; but even in this case the intestinal phthisis may take such a predominance that all attention is turned to, and should be fixed on, the intestinal lesions which by themselves compromise the life of the patient. This is so true, that in the opinion of certain physicians there is a sort of alternation between the intestinal affection and the state of the lung; the intestine assuming all the morbid process as opposed to the progress of the pulmonary malady. Unhappily there is here only a deceitful appearance during, the fatal evolution

of intestinal phthisis, if the pulmonary symptoms seem to diminish, if the cough and the dyspnœa are restored to relative calm, the pulmonary lesion does not proceed less fatally; auscultation and percussion are available to show the progress in destruction of tissues, and their evidence is unchallengeable. The parallel state of the two organs is complete from an anatomical point of view, only the phenomena expressed are imperfect on one side or the other. However it may be, in the actual state of our knowledge, where tuberculosis of intestine is secondary, we are right to suppose that it results from the infection of intestine by tubercular *crachats* which have been swallowed by the patient.

(b) *The intestinal lesion may be primordial.*—If intestinal phthisis is often secondary and even tardy, it is not less true that it may appear before pulmonary phthisis, and become itself the starting-point of the diffusion of tubercles, that is, the propagation of the bacilli to other organs. It is with children especially that we find this localized tuberculosis of abdominal organs, which commences generally by the intestine, afterwards to gain the peritoneum. We may, by good right, suppose that in this case the infection takes place in the intestine itself, and that the tuberculosis has penetrated into the economy by this way, that is, by the introduction of bacilliferous food, particularly of milk from tubercular cows. This grave question of preventive hygiene has been already raised in discussing the origin of tuberculisation.

Anatomical characters and seat of intestinal tuberculosis.—The intestinal tubercle, identical with that of other mucous membranes, has its habitual point of departure in the lymphatic apparatus; that is to say, in the solitary and agminated glands (Brunner's glands and Peyer's patches). We find, under the epithelium of the mucous membranes and

villi, the first miliary tubercles, which rarely remain in this state (a third of the cases) (Louis). In general, they do not delay in forming a vast tubercular infiltration, which compresses the tubular glands and villi. At a more advanced period the infiltration gains in depth and extent, so as to reach the sub-mucous tissue, the muscular layer, and the serous covering; at the same time, the neoplasm gains the surface, and, spreading from place to place, constitutes tuberculous ulcerations, of which some increase transversely under the form of rings following the vascular territory of the intestine. The rest generally parallel amongst themselves, and perpendicular to the long axis of the intestine (Andral, Rindfleisch, Lancereaux, Laveran).

In the large intestine we find, as in the small intestine, follicles which have undergone suppuration or ulceration, and are often fused into ulcers of oval form with detached, jagged edges, but without precise direction. The lesions are analogous to those of dysentery; that is, diffuse lesions occupying the last portions of large intestine in the form of ecchymotic tumefaction, and enormous anfractuous ulcers, amongst which one finds here and there certain ulcerated follicles. In the whole of the intestinal tube the lymphatic system is attacked throughout without delay; the mesenteric glands often to such an extent that Colin demands if they have not been the starting point of the ulcers in the mucous membrane.

V. *Diagnosis of catarrhal, nervo-motor, ulcero-tuberculous, and amyloid diarrhœas.*—The symptoms common to all these forms of intestinal disease are diarrhœa and abdominal pains, or colics. Louis does not attach any diagnostic importance to the nature, intensity, or seat of the pains; it is only by the number of evacuations and their composition that we must be guided in deciding the differences which separate premonitory and pretubercular diarrhœas

from those which depend on ulceration or colliquative flux. It is important, then, to indicate briefly the physiological state of evacuated materials, as a point of comparison with the modifications that anatomical lesions impress on them, as well as nervo-motor troubles of intestine.

(a) *Histochemistry of alvine materials in a physiological state.*—When the contents of the intestine are put in motion with such quickness that they abandon the digestive tube before the alimentary substances and the liquids secreted for their digestion have had time to undergo the regular transformation, especially before the principles resulting from their digestion have been given up to absorption by the lymphatic vessels and the portal system, then it is that diarrhœa is produced (Traube). In the normal condition the residues of digestion, before being eliminated, sojourn a sufficient time at the end of the large intestine in the sigmoid flexure of colon and rectum, as proved by the fact that evacuation takes place but once or twice a day, in spite of the multiplicity of our repasts; percussion over the left iliac fossa is also sufficient to show the presence of fæcal masses there. It is this time of detention which enables the stercoral matter to become more consistent, a great part of the water being reabsorbed by the intestine. In fact, the intestinal contents then show less water, less cholalic acid, no traces of the twin biliary acids (glycocholic and taurocholic); bilirubin, bilifuscin, that is, the bile pigments which give Gmelin's reaction, exist no longer, but are replaced by a darker colour, there are some vestiges of soda salts, mucin, and fat, and that is all. Suppose, now, that the evacuations should be premature, they will be more watery, more numerous, in general more decolourized or more *yellow* than normal fæcal matters. They contain, then, a great quantity of soluble salts, cholalic acid, albumen, fat, mucin, and further a certain number of

substances which are normally found but in the small intestine as the twin biliary acids and bilirubin; in a word, they approach the more closely to the composition of the contents of small intestine the more quickly they have been eliminated. With the microscope we find, even in the normal state, (1) cylindrical epithelial cells, which have often become granular or swollen (Nothnagel); (2) some leucocytes; (3) crystals of ammoniaco-magnesian phosphate, cholesterin, fatty acids, and bilirubin; (4) as a constant fact, the presence of all sorts of bacteria, and even the bacillus subtilis, so that we cannot, according to Nothnagel, argue from that in concluding that there exists catarrhal irritation of specific or parasitic origin.

VI. *Chemistry of pathological diarrhœal stools. Diarrhœa of nervo-motor origin.*—There is no diarrhœa without the nervo-motor element of intestine being brought into play; diarrhœa is never provoked but by sensory irritation of intestinal mucous membrane, that is, the terminal extremities of sympathetic nerves, which impression is propagated to the medullary centre, or simply to the intrinsic ganglia of intestine (Meissner's especially), to be reflected to the muscular coats of intestine, and determine, especially in the large intestine, exaggerated peristaltic contractions. This reflex excitant may be of nervous origin, and constitute all the malady. It is not this kind of nervous diarrhœa which attacks consumptives; but there is no acute or chronic catarrh, simple or follicular ulceration, or even tubercular ulceration, which can augment the intestinal peristaltic action without exciting these terminal nerve-endings. Evacuations increased in number have no other cause; only the normal contents of the intestinal tube come to join the morbid product, the more or less abundant and diversely constituted exudation when we have to deal with catarrhal or ulcerous inflammation.

(b) *Diarrhœa of catarrhal origin.*—Intestinal catarrh is rarely limited to the small intestine without the cæcum and the colon participating. If the irritation had been confined to the duodenum, we should not even have diarrhœa, which one knows is due nearly exclusively to the contractions of large intestine. When the catarrh occupies a great part of the small intestine, the materials, without being diarrhœal, may nevertheless show their catarrhal origin; they are, according to Nothnagel, intimately mixed with small hyaline fragments of mucus, which can only be recognized by the microscope. As soon as the irritation has invaded the first portions of large intestine, we find in the alvine matters certain elements which come exclusively from the small intestine, and which in the normal condition always disappear in the colon. We must note specially: (1) the remains of undigested food; (2) especially a large quantity of muscular fibres, often even visible to the naked eye, which constitutes lienteric diarrhœa; (3) particles of starch and fat. All these elements are mixed with extremely liquid material, which contains up to 90 and 95 per cent. of water, instead of the normal proportion of 75 per cent. What characterizes in a definite manner the catarrh of jejunum and ileum, is the presence of bile. In the normal condition it is only the contents of small intestine which give Gmelin's reaction; the materials of large intestine, that is to say, normal fæcal matters, do not furnish traces of it. For bile to appear with its colouring matter undecomposed, the peristaltic action must be augmented throughout the digestive tube. We know that with children the stools described as green, and which are considered dangerous, come precisely from this undecomposed bile; in reality, they give with nitric acid the characteristic colour reaction. In other cases of enteritis of small intestine, we find bile, colouring certain portions of

stercoral matter, besides the mucous corpuscles and cylindrical epithelium coloured yellow. Lastly, it is to be noted that these green materials do not often become so but on exposure to the air. At the first moment they appear yellow; it is biliverdin which is transformed by oxidation, and takes a green colour. Catarrh, or at least peristole of large intestine, accompanies nearly all diarrhœas; we cannot explain without that the abundance and tenuity of the stools. What especially characterizes the participation of the colon in the irritation is the presence of easily recognizable masses of mucus; whilst in irritation of jejunum and ileum the mucus is intimately mixed with other excrementitious elements, and is recognized but with the microscope, colitis is known by superposed mucous layers surrounding the fæces. In inflammation of the lower part of large intestine the diarrhœal materials often take a dysenteric form; it is pure mucus, mixed with pus in varying quantity. All these distinctions, based on the localization of diarrhœas, present considerable interest from a therapeutic point of view, more still with regard to the diagnosis of catarrhal and ulcerous diarrhœas.

(c) *Ulcerous diarrhœas.*—These are distinguished, according to Louis, by their indefinite duration and by the frequency of their evacuations, so that one may in a tubercular patient suspect ulceration when such evacuations last for more than six weeks; whilst, further, they contain blood, or are of a particular red colour, and lastly present the putrid odour of macerated tissues. The dysenteric condition of stools is never found with catarrhal evacuations; for a stronger reason never the hæmorrhage which is to be found in certain cases (Laveran, Bernheim).

(d) *Colliquative diarrhœa by amyloid degeneration.*—It is true that certain characters, amongst others the red colouration, belong also to colliquative diarrhœa, resulting from

the amyloid degeneration of intestinal arteries. What distinguishes these diarrhœas is: (1) Absence of abdominal pain and sensibility even when the flux is considerable (Traube); (2) the absence of intestinal hæmorrhage or blood in the evacuations—the microscope does not allow us even to recognize red globules in the midst of the whitish liquid which constitutes the last form of diarrhœa; (3) the coincidence of special diarrhœa with other amyloid degenerations, in general the order of invasion of organs by this amyloid transformation, is this: the liver first, afterwards the spleen, and kidneys, lastly the intestinal mucous membrane, so that if the urine is free from albumen, if dropsy is absent, we may decide against colliquative diarrhœa.

(e) *Résumé.*—After these data the law of Louis may be modified and thus formulated: a prolonged intense diarrhœa, which is manifested in the course of pulmonary tuberculosis, should make us believe in ulceration if the diet of the *invalid* has not been altered, if the diarrhœa is characterized by sanguinolent evacuations, at the same time by colic, great abdominal sensibility and no traces of amyloid degeneration in any organ. On the other hand, we may affirm that the case is but that of catarrhal diarrhœa, in the absence of indicated conditions, even when the evacuations persist and resist the most moderate regimen.

(f) *Meteorism and gurgling.*—Meteorism exists in all these cases; percussion shows only that the sigmoid flexure contains more gas than normally; palpation demonstrates a gurgling sound in the ileo-cæcal region; pressure causes pain and augments the sensibility of abdomen by displacing gas, but none of these phenomena belong exclusively to a special kind of diarrhœa; pain alone appears to exclude amyloid degeneration.

VII. *Ulcero-tubercular diarrhœa in the child.*—We have

said with reason that primitive tuberculosis of the intestine is common in the child. People have accused bacillary milk coming from affected cows; for that the child, to become ill, must have been brought up with the bottle. The rarity of tuberculisation in children before the age of two years is an argument against lacto-bacillary infection. Phthisis begins to be fatal between two and five years, and more still from five to ten, which is a time when the child does not take relatively more milk than the adult. During all periods of child-life intestinal phthisis predominates over pulmonary phthisis in such fashion that tubercle may, after having attacked the intestine, gain the peritoneum, lymphatic glands, liver, etc., the lungs remaining perfectly free. The characters of *tabes mesenterica* (called also *carreau*, on account of the tubercular engorgement of mesenteric glands) are thus made evident: (1) Mucous or lienteric diarrhœa, often dysenteric (rarely bilious), which resists all methods of treatment; (2) meteorism so pronounced as to distend the whole abdominal wall—it is not till the end that the belly is flaccid and flattened; (3) spontaneous pains, colics—they depend ordinarily on the accumulation of gas, and are temporarily relieved by the escape of these gases, which are fœtid, showing they come from decomposition of fæcal matters; (4) the sensibility of the belly results frequently from the participation of the peritoneum in the tuberculosis; (5) a frightful wasting, with the most intense anæmia, which is often out of all proportion to the intestinal losses, manifest themselves from the start.

VIII. *Diagnosis of ulcerous diarrhœa and diarrhœa of dyspeptic origin in the child.*—Tuberculous tabes being rare in children below the age of two years, it is necessary only to distinguish simple chronic catarrh, which is manifested in the child at all ages. In most cases it depends on defective or insufficient nourishment; thus food too ex-

clusively starchy, especially if albuminoid materials, such as meat, are deficient, is enough to produce gastro-intestinal irritation. In reality these foods are badly digested, incompletely peptonized, and for that alone they decompose in the intestine. The products of putrefaction produce a mucous secretion, and at the same time excite muscular contractions. Thus dyspepsia determines a catarrh, which in its turn, and by a sort of vicious circle, augments still more the difficulties of digestion. The distinction between dyspepsia and catarrh exists so much the less for that in reality we have to deal with dyspepsia at once alimentary and mucous. Therefore this grave condition is characterized especially by more numerous, more watery stools, containing particles of food which have escaped digestion, quantities of mucus often agglomerated like grains of sago, leucocytes, epithelia, rarely blood. These characters are found in a more marked manner in intestinal phthisis, which is accompanied especially by severe pains, fever, and anæmia, which are not found in gastro-intestinal dyspepsia. The wasting which is observed following all diarrhœal affections is much more early, more considerable, in *tabes mesenterica* than catarrhal enteritis. There is a real importance in establishing the diagnosis between an incurable lesion and a dyspepsia, which, despite all its gravity, may yield considerably to change of diet and better-regulated hygienic conditions.

§ 105. *Intestinal Bacilli.*

Following Koch and other authors, the presence of the *specific* bacillus may be regarded as a certain sign of the tubercular nature of diarrhœa.

§ 106. *Tuberculo-Bacillary Affections of Kidneys.*

Localization.—The kidneys certainly eliminate bacilli, as also the other microphytes contained in the body.

Benda has found them principally in the Malpighian glomeruli, where they form non-nodular tissue, distinct from tubercle and of irritant nature. In the uriniferous tubes they produce but insignificant lesions.

Urine.—The urine frequently contains bacteria. Bouchard has described bacterian albuminuria. The bacillus itself is also to be found, which indicates an invasion of renal or vesical tissue, or perhaps the elimination of parasites coming from the other organs. We can by this necessary examination, which presents, however, certain difficulties, unmask phthisis when it is primordially localized in the urinary system.

§ 107. *Lesions of Organs.*

I. *Genital organs of man. Tubercles of testicle.*—Tubercles of testicle constitute a type of local and primitive tuberculosis. One sees often with the adult, tubercles produced in the testicles, which in no way influence the lungs. Two-thirds of the tumours of this kind are localized and remain so (Reclus). It is not altogether impossible that tuberculous infection may start from this organ, and phthisis itself may come to end the morbid local process. Developed phthisis may have its influence on the testicle, commencing round the seminal tubes (Malassez).

On the other hand, the prostate may equally become the seat of granulations, or even caseous infiltrations, which may terminate by veritable excavations.

II. *Genital organs of the woman. Uterus. Metritis and tuberculosis.*—Catarrhal metritis is so frequent in consumptives, and the loss which ensues from the uterine leucorrhœa contributes not a little to weaken the patients, so much so that a well-known physician has ended by treating them almost exclusively for the uterine affection.

Tubercles.—Tuberculous lesions are developed, especially

in the mucous membrane, and may be carried to such a pitch that the cavity of the uterus is found filled with caseous masses. In the cervix the tubercles are more rare; but granulations have been observed in the ovaries of phthisical patients (Raynaud, Siredey).

§ 108. *Tubercular Peritonitis.*

The peritoneum is very often the seat of primitive tuberculosis, in the same way as by the implantations of bacillo-tuberculosis material in the peritoneum. The penetration of bacilli into the serous cavity of the abdomen does not delay long in being followed by extended disseminated tuberculosis, a true granular affection of all the membrane. It is one of the most grave modes of infection; all the serous membranes take it with this facility and intensity. The peritoneum is affected with tubercle also after intestinal ulcerations, following bacillary infection. The disease has yet another mode of propagation. This is the tubercular and inflammatory process in the pleura, which attacks the peritoneum by the lymphatic channels through the diaphragm.

FOURTH CATEGORY.

Doubtful and Advanced Phthisis and Pseudo-Phthisis.

XXXIII.

SEMEIOLOGY OF INDURATED AND CAVITARY PHTHISIS.

(a) *Local signs.*—Errors may arise from the simple fact that *all* induration, *all* cavities, resemble those of tuberculous nature.

(b) *General signs of oxidations.*—For stronger reasons

we cannot help in elucidating the diagnosis by general reactions, oxidations, alterations of blood, and urine, fever, cardiac, and nervous troubles; in a word, all that indicates the invasion of the entire organism, all that one usually calls vaguely *general condition.* But if this study is a little sterile from this point of view, it is of capital importance with regard to prognosis, which is governed solely by the gravity or benignity of accidents and lesions which result from the struggle of the organism against the parasite; of the *struggle for life.*

(c) *Lesions of denutrition and tuberculization of organs.*—The lesions of different orders which are produced in organs invaded by the parasite. It is not solely tubercle which results from the action of the bacillus. There are also apparently simple inflammations, fatty degenerations, amyloid transformations; hæmorrhage with infarctus, thrombosis, emboli, or dropsy; failure everywhere, denutrition in all its forms, in all organs. Here is the balance sheet of phthisis.

The pathological scale commences by functional trouble, and finishes by the most grave anatomical alterations. The same pathological series is found for all the organs, the heart and its coverings, for the kidneys, liver, spleen, for the nervous system. Everywhere lesions of organs are linked to chemical changes, that is oxidation.

§ 109. *Cardiac and Febrile Phenomena. Lesions of Heart and Pericardium.*

Phthisis is rarely apyretic from one end to the other. It often even begins with troubles of temperature and circulation.

I. *Pulse.*—Frequency of pulse, in a great number of phthisical patients, is not correlative with the fever; it is often found in apyretic tuberculous sufferers. They do

not, however, perceive this acceleration in the beating of heart and pulse; whilst it is produced and exaggerated under the influence of the least causes, the least physical exercise, moral impression, or most moderate repast.

II. *Cardiac palpitations.*—The greater number of invalids notice this increased action of heart, and complain of palpitations, nearly always accompanied by a certain amount of distress, although not painful. Peter has with reason insisted on the importance of this strange phenomenon, which is so often attached, not as people say, by chance, to a fatal susceptibility, but to the early weakness of the *future patient.* It is difficult not to consider these palpitations as an index of the diminution in excitability of cardiac vagus nerves. We know that the palpitations cannot be explained by irritation of sympathetic, or of the acceleratory, nerves. They are phenomena of a paralytic order; symptoms of exhaustion in the moderator or the inhibitory nerves. It is little noticed that the heart is thus abandoned to itself without curb; of small importance that it produces, at the same time, an anxiety which resembles dyspnœa. The patients, and especially medical men, mistake the nature of the evil, and attend only to the cardiac manifestation, without occupying themselves with the respiratory organs.

III. *Heart. Volume of the heart.*—However, at this time the volume of the heart is in no way augmented. There is neither hypertrophy nor cardiac dilatation at this period of the malady. Sometimes even the præcardial dulness appears diminished.

Diffusion and intensity of cardiac sounds.—In general we find a murmur during the systole, by preference at the arterial orifices. What is its signification? Have we to deal with endocarditis, an anæmic murmur, or a nervo-motor *souffle?* Endocarditis has infallible signs which

denote the localization of the inflammation, and especially of morbid products on the borders of valves or orifices. They are persistent valvular murmurs, fixed at one or the other orifice, with one or the other sound. Anæmia does not fail to be shown by vascular murmurs when it is pronounced at the cardiac region. We have to deal with a nervo-muscular murmur, which is ordinarily at two orifices, very variable, very fugitive, which an increased flow of blood infallibly augments, and which the calm of circulation makes disappear. Do not be afraid of this murmur, however intense it be. It must be associated with other signs to indicate a lesion of myocardium or valves.

IV. *Lesions of heart and pericardium.*—Phthisis sometimes acts on the heart and pericardium in a manner to place its lesions in the first rank; it seems, then, that pulmonary tuberculosis takes the cardiac form.

Persistent palpitations.—Already we have indicated a danger to be avoided in the beginning, when the lung has not yet been ostensibly attacked, and the patient is a prey to those palpitations which constitute a real delirium of the heart. The difficulty of avoiding a mistake is much more marked when inflammatory or degenerative lesions invade the parts constituting the organ of circulation. We must note under this head: (*a*) myocarditis, or fatty and fibrous degeneration of cardiac muscle; (*b*) vegetative endocarditis of valves; (*c*) simple pericarditis, tubercular pericarditis which come from the pleura by way of propagation; (*d*) in the forms accompanied by dyspnœa, or very extensive tuberculosis, the right heart does not fail to hypertrophy or to dilate.

(a) *Degeneration of myocardium.*—Myocarditis, or rather sclerosis of heart, is not shown by any certain sign, any direct trouble in the action of the heart; there are neither enfeebled nor exaggerated cardiac sounds. Sometimes there

is an arhythmic condition, also a systolic murmur may exist. It is at most when the heart is dilated as well as degenerated that the increased dulness explains the change of capacity. The real and, so to speak, constant signs of degeneration of myocardium are shown on the side of respiration only, by dyspnœa either by fits or continuously, and it is then that the difficult problem of resolving the pulmonary or cardiac origin of the dyspnœa is presented. I have reason to say that phthisis is sometimes masked by the heart.

(b) *Vegetative endocarditis.*—When tuberculosis attacks the endocardium it is rare that the evil is mistaken. Endocarditis attacking the valves determines valvular insufficiency or retraction of orifice, which is shown by one or the other of certain signs, particularly by local circumscribed murmurs corresponding to the altered orifices.

(c) *Dry pericarditis.*—When the pericardium is attacked under the form of plastic pericarditis, the friction is unmistakable evidence of this lesion, which must be looked for because it does not determine any appreciable trouble which draws attention to it.

(d) *Pericarditis with effusion.*—It is not the same with pericardial effusion, which is manifested by cardiac signs at the same time as perturbation in functions of lung and peripheral circulation.

(e) *Tuberculosis of pericardium.*—This is usually propagated from the pleura, and is relatively frequent. The effusion is often abundant (one to two litres), and easily becomes hæmorrhagic.

(f) *Dilatation of right heart. Tricuspid insufficiency.*—Following dyspnœa, pulmonary congestion, the invasion of the whole respiratory tract by tubercles, the right heart ends by dilating. There ensues a relative tricuspid insufficiency which is shown by hepatic or renal congestion, swelling of lower limbs. The consumptives who die

through the heart are especially those who have slowly developed phthisis of fibrous and emphysematous form.

§ 110A. *Fever.*

The thermometer indicates the course of the fever. One may decide by the temperature chart that a fever, said to be intermittent, has no regular periods; that it is not manifested every day at exactly the same hour; that it is never of the tertian kind. On the contrary, it is rather remittent, and the intervals of access are never completely apyretic. The examination of the chest shows in most cases that phthisis is already in the second stage, which is to be recognized by foci of dry sub-crepitant râles or cracklings with marked tympanism; in reality, with intense persistent fever, it is rare that tuberculous nodules do not rapidly become caseous and undergo fatty liquefaction. If it is not so, if softening is not evident, we may be sure that the tubercular infiltration is sufficient to give rise to modifications of respiratory system, particularly prolonged expiration. Of course the sure method of examining the *crachats* may be employed for diagnostic purposes in these cases. The fever may be of various types.

§ 110B. *Diverse Phenomena of Nutrition.*

Wasting, deformity of phalanges and nails must be noted, also the pale colour of skin, and the eruptions of *Chloasma phthisicorum and Pityriasis versicolor.*

§ 111A. § 111B. *Urine.*

It has been clearly shown that consumptives lose phosphates by the urine (Teissier) as well as by the expectoration (Daremberg); the diminution in body weight, corresponds to the augmentation of calcic phosphates in urine. Phosphates diminish in urine after the ingestion of

fatty materials and hydrocarbons; cod-liver oil, without any doubt, reduces the phosphatic loss (Hanot); it is the same with feculent substances which are so useful to phthisical patients. The quantity of phosphates lost is raised to three or four grammes per litre of urine, but only at the start of the malady; when it comes to the period of cachexia the loss of phosphates is stopped (Teissier). Daremberg has shown the correlation of phosphaturia with phosphatic expectoration. The same reflexions apply to loss of chlorides which are eliminated in notable quantity by the urine, and more still in the expectoration, except there comes pneumonia.

Diazotic urine. Application to diagnosis.—Diazotic compounds which result from the action of nitrous acid on the amides of the aromatic series are distinguished by special colouring power. Ehrlich uses the sulphanilic acid of commerce. In a flask containing half a litre of water a solution of sulphanilic acid is made, from 40 to 50^{cc} of nitrous acid, and in solution a few grains of nitrate of soda, are added. To show the reaction an equal quantity of urine is added to 1^{cc} of reagent; this is saturated with ammonia and let stand for twenty-four hours. Normal urine remains unchanged, but with the urine of consumptives, and most fever patients, we see a scarlet or orange-red colour appear. Later this red colouring material is dissolved and transformed into an insoluble green body which colours, as billiard-cloth-green, the superficial layer of sediment. This reaction is most constant in phthisis, typhoid fever, measles, and typhus; it is always wanting in pneumonia or diphtheria.

In the actual state of science it is impossible to disregard acquired facts; it will not be rash to give this reaction a diagnostic, and especially a prognostic, value. The intensity of the colouration is in general a guide to the importance of the malady.

Kidneys.—Parenchymatous nephritis is a complication

of all cachexias, and is known by albuminuria, often by dropsy. Interstitial nephritis is rarely recognized during life. Amyloid degeneration is found in two-thirds of the phthisical patients who have reached the period of ulceration. It ordinarily coincides with amyloid degeneration of liver, spleen, and vessels. It is generally revealed, though not in a constant manner, by albuminuria, sometimes by dropsy of lower limbs. Tuberculosis of the kidneys, which is generally secondary, attacks by preference the cortical substance around the uriniferous tubules.

§ 112. *Blood. Hæmapoietic Glands. Vessels.*

Blood. (a) *Pretubercular globular anæmia.*—This is often one of the revealing indices of the malady. Anæmia or pseudo-chlorosis requires a special examination; there is either a diminution in number of globules or of hæmoglobin.

(b) *Tardy anæmia.*—What is certain is that the number of red blood globules in man falls from five or six millions per cubic millimeter to two and a half millions, and in woman to less than one million; the leucocytes, on the contrary, augment in number.

(c) *Anæmia by loss.*—Intestinal catarrh, and bronchial catarrh, causing the loss of blood-cells, mucus, epithelia, and phosphates, contribute singularly in favouring the production of these *total* anæmias which are terminated by diminution of all plasmatic elements of blood.

Liver.—The liver always presents alterations, some of which are grave. Fatty degeneration, or more often infiltration, is found in 67 per cent. of the cases dying from phthisis (Frerichs); hyperæmia, which is often combined with this last lesion to constitute the nutmeg liver, and amyloid transformation are often enough observed. Tubercles, which may be developed at all periods of the malady, are not known by any clinical sign. As hepatic

tuberculosis often coincides with that of the intestine, we suppose that the liver affection is secondary, and that the bacillus has penetrated by way of absorption from the intestine into the vena porta and liver.

Spleen.—The spleen is also subject, like the liver, to blood stasis, amyloid degeneration, and tuberculosis, which is presented there under the form of caseous foci. In all these cases there is an augmentation in the volume of the organ.

Vessels. Lesion of circulation.—Under this head I comprehend: (*a*) Thrombosis of pulmonary artery, which plays an important part in the production of ultimate accidents. (*b*) Phlegmasia alba dolens, which is one of the most grave signs of circulatory trouble. (*c*) Dropsies, which may have their starting-point in the renal lesion (which is rare), or in obliterations of the veins. (*d*) Hæmorrhage, which is observed often under most varied forms and in the most dissimilar tissues, even in the skin and subcutaneous cellular tissue under the form of purpura.

§ 113. *Functional Troubles and Lesions of Nervo-muscular System.*

These are (*a*) sensory troubles, pains in upper limbs, sternum, back, and sometimes over sciatic regions. (*b*) Troubles of muscular sensibility and muscular contractions, over-excitability of muscles, functional troubles of cerebro-spinal centres, such as hypochondriasis, initial delirium, mental alienation, and final delirium, cerebro-spinal lesions of inflammatory or tubercular nature, simple meningitis, tubercular meningitis, and granulations. If one sees headache with vomiting, constipation, contraction of the muscles of the neck, partial paralysis of the face, vaso-motor troubles, dilatation of pupils, and especially softening as well as irregularity of pulse, come on in the

child or the adult, before or after pulmonary phenomena, we can be certain there is formed, at the base of the cranium, near the bulb, an exudation which compresses and excites the origin of vagus nerve and vaso-motor centre. The branches of the vagus destined for the stomach determine vomiting by their irritation; the over-excited cardiac branches diminish the contractions of the heart; the irritated vaso-motor centre produces the famous *tache méningitique*. Meningitis causes death, whether the lung be intact or damaged. It is often complicated by tubercular spinal meningitis, which is characterized principally by contractions of muscles of trunk and members. Pachymeningitis, encephalitis, cerebral anæmia, are also found frequently; but hyperæmia is much more rare. Hæmorrhage, softening, cerebral tubercles, chronic meningo-myelitis, tubercles of spinal cord, have all frequently been found in tubercular patients.

§ 114. *Anatomical Characters and Semeiology of Fibrous Phthisis.*

The lung is subject to a fibrous interstitial process; the pleura is thickened and transformed into fibrous tissue, which often forms adhesions with pericardium. The displacement of the apex of the heart and area of cardiac dulness is to be noted; this is usually towards the side affected, which is strongly retracted; the depressions and intercostal spaces are deeper. The clinical symptoms recall, in all respects, those of bronchial dilatation, complicated with pulmonary cirrhosis, and the crises represent all the phenomena of acute asthma grafted on to permanent emphysema. Ducastel declares the *crachats* do not contain bacilli, in which case I say the diagnosis is impossible.

XXXIV.

DIAGNOSIS OF ADVANCED AND DOUBTFUL PHTHISIS, ALSO PSEUDO-PHTHISIS.

HERE is a list of maladies which may simulate phthisis: —

1. Asthma and emphysema.
2. Dilatation of bronchi.
3. Engorgement of bronchial glands.
4. Syphilis of lung.
5. Pulmonary cancer.
6. Hydatid cysts.
7. Pneumothorax.

Inversely, this fourth categorical list of phthisis comprises conditions which may simulate other maladies, such as (*a*) massive indurations of lung; (*b*) excavations of all other kinds; (*c*) bronchial catarrh and asthma. In most of these cases our best and only diagnostic method is microscopic examination of expectoration.

§ 115. *Emphysematous Phthisis.*

Emphysema may be mechanical, a fault of nutrition, or caused by paralysis of pneumogastric nerves. In the development of mechanical emphysema, it is always loss of elasticity which plays the principal part, and that during both inspiration and expiration, more particularly the latter.

§ 116. *Ganglio-bronchial Tuberculosis.*

The anatomy and pathology of this affection have been well described by Gouguenheim.

XXXV.

GENERAL ACUTE PHTHISIS.

In the midst of perfect health an individual is taken with initial shivering, soon followed by frequent, spasmodic, fatiguing, dry cough, exactly like localized pulmonary phthisis at its start. At the same time the respiration becomes difficult; often there is considerable dyspnœa, aggravated persistently by true fits of suffocation.

Fever.—The fever, at first moderate enough, and nearly always accompanied by night-sweats, does not present any well-characterized thermometric curves; we see irregular oscillations from time to time, a leap or ascension (Lépine), of which it is often difficult to divine the origin.

Percussion.—The physical signs indicate condensation of pulmonary tissue. The percussion sound presented at the level of the tubercular masses has a tympanic character; but there may be absolute dulness when the tubercular infiltration is superficial enough and thick enough to push back the lung. Traube places the lesion in the anterior summit of a single lung, but Hérard and Cornil, as well as Lépine, think the mischief starts from below.

Auscultation.—Sub-crepitant *râles*, more or less fine, and dry, fixed at the same place, are replaced at the end of from eight to fifteen days by a bronchial *souffle;* these are the signs of a tuberculo-caseous group, which is generally found at the apex of the lung; sub-crepitant *râles*, concentrated especially in upper lobes, then cavernulous *râles*, amphoric *souffle*. Later, the same series is repeated in lower regions, and predominates so that we forget the cavernulous *râles* of upper lobes.

General signs.—Want of appetite, dryness of tongue, tension of belly, sometimes diarrhœa, complete this picture of amphorism, and contribute, with rapid febrile consumption of tissues, to precipitate a fatal termination.

Diagnosis.—There is nothing special as to the diagnosis. Reunite the series of phenomena in the period of induration and ulceration of chronic phthisis, and you will have established the basis of diagnosis of this very rapid ulcerative destruction of lungs; nothing can obscure the opinion of the practitioner as to this matter.

Course of the malady.—In fifteen or twenty days the patients have already lost flesh, partly from loss of appetite, and often under the influence of diarrhœa. Most die from rapid consumption; it is amongst these that one immediately finds the bacillus.

XXXVI.

ACUTE MILIARY PHTHISIS.

This is infectious bacillary phthisis, which invades all the organism in a rapid and fatal manner; and that is the only point of contact with ordinary phthisis of acute course or pneumonic form.

Definition from anatomical and ætiological Characters.—A few gray granulations are not enough to characterize miliary phthisis; once more the lesion has no value but by the action of the specific cause, the bacillus; phthisis exists, but at the price of general parasitic infection. We characterize the granulation; because it habitually preserves its texture and primitive aspect, it has been long separated from the granulation of properly called tubercle. Whilst Bayle considered granular phthisis as distinct from tubercular phthisis, Empis made of it a foreign, febrile phlegmasy in the group of tuberculosis, and Robin assigned to

the granulation, the property of not becoming caseous like tubercle. Virchow, on the contrary, taught it to be the unique, true, and only tubercle, and that all that became caseous was within the domain of degenerative inflammation. The truth is, that the granulation of acute miliary tuberculosis, having arrived at a certain degree of development, may lose its transparence, and present in the central part evident traces of caseous transformation. The question of anatomical identity has been considered, and the question of bacillary origin has been solved.

Diffusion of miliary tuberculosis.—What distinguishes it most easily from common phthisis is its tendency to generalization. It is in reality rare to see it rest circumscribed in a single organ as the lungs, or in a single tissue as the serous membranes. Ordinarily it invades organs in the following order of frequency: lungs, 76 per cent.; liver, 82 per cent.; spleen, where the granulations are sometimes hardly visible to the naked eye, 57 per cent.; the kidneys figure for 62 per cent.; intestines, 56 per cent. Though less frequently attacked than these organs, we must mention the choroid, which, examined by the ophthalmoscope, often reveals a doubtful or latent malady. Then come, in a quarter of the cases, the peritoneal and pleural, serous membranes, the meninges, pia mater and dura mater, which are nearly always the seat of grave inflammations of granular origin; the other organs, as the brain, heart, bones, female genital organs, are noted only by exception in the post-mortem records of Simmonds. Lastly, it is remarkable that the salivary glands and pancreas completely escape a general law. It is admitted that acute miliary phthisis may present the characters of an infectious or auto-infectious malady, or simply the course of a broncho-pulmonary affection. The ophthalmoscope is very useful for deciding a doubtful diagnosis.

§ 117. *First Group. Generalized Infectious Tuberculosis.*

As all other infectious maladies, especially typhoid fever, miliary phthisis is known by (1) nervo-muscular troubles; (2) by fever; (3) abnormal state of digestive organs, secretions, blood, spleen; (4) often by alterations in nutrition and tegumentary circulation. The most important signs are the rapid wasting and paleness of the consumptive patient, and the irregular fever. The history of previous tubercular or scrofulous lesions must be carefully inquired into; we have often to deal with a true auto-infection.

§ 118A. *Second Group. Miliary Tuberculosis localized in Respiratory Organs.*

We find four principal forms: tuberculosis of bronchi, bronchioles, lungs, or pleura. These are designated as catarrhal, capillary, asphyxial, and pleural.

Miliary phthisis. Bronchial tuberculosis.—We have three signs which rivet attention, sometimes from the first appearance of the bronchitis, but always later. 1. Dyspnœa, which is never found in simple bronchitis, with the following characters: the respiration is generally accelerated to such a pitch that in a child or even an adult attacked by miliary bronchial tuberculosis we may count forty or even sixty respirations in a minute. At the same time the respiration is extremely profound, often whistling, always painful, and accompanied by real anguish. This oppression is continued or is manifested under the form of prolonged attacks; but what is most remarkable is the discordance between these forced inspirations and the insignificant objective phenomena, that is, the results of auscultation and percussion.

2. *Cyanotic aspect of patient* who is pale, and with cyanosis most marked on lips.

3. *Suppression of respiratory murmur and localization*

of râles. And, in addition, the general signs of tuberculosis must be taken into account.

Capillary bronchitis. Miliary tuberculosis of bronchioles.—The diagnosis is nearly impossible; as Jaccoud says, we must rely on the signs of general tuberculosis (fever, wasting), or the coincidence of caseous centres in some other part of the body, especially the pleura.

Miliary pulmonary tuberculosis.—This has been long perfectly described. It is most frequent from two to five years; more rare in later life, but found sometimes in the aged. It is characterized by sudden, violent, ordinarily continuous dyspnœa, augmenting from day to day until the final asphyxia. There is nothing to be found on percussion, for the lung is sound in the intervals of granulation; no *râles,* for there is no reaction; but there is enfeebling of respiratory murmur from obstruction. Instead of the long forced whistling expirations of asthma, it is inspiration which necessitates the employment of all auxiliary forces by the patient; it seems that the air cannot penetrate into lungs crammed with tubercles.

§ 118B. *The Parasitic Origin of the Malady.*

This has been established in the same satisfactory and complete manner as that of ordinary tubercle.

Pleural miliary phthisis.—See article "Pleurisy," § 101.

§ 119. *Phthisis of Children.*

Comparison with phthisis of adults.

We may thus characterize the phthisis of childhood from five to fifteen years.

1. Acute generalized miliary phthisis is that which dominates all others; it is often grafted on to foci of scrofulous glands, whether cervical or bronchial. 2. In its generalization the granular condition may damage the cerebro-spinal

meninges and thus produce death. 3. But it is not rare to see the granulation invade the lungs and the pleura in a manner to simulate, during life, bronchitis or acute bronchiolitis and suffocating catarrh. 4. Phthisis localized in the lung, or pneumonic phthisis, is rare, and never takes a chronic form. It is a true tuberculous broncho-pneumonia which is developed in a few weeks as granulations, or in a few months as the caseo-tuberculous pneumonia of the adult. 5. The phenomena are entirely comprehended in the signs of caseous induration, and ulceration, that is dulness, bronchial *souffle*, bronchophony, and resonant *râles;* to these physical signs we must add fever and wasting, which follow its acute course. 6. Tuberculisation of bronchial glands is frequent.

Phthisis of old people.—We have no exact statistics of deaths amongst the aged, but from sixty to seventy is the period of greatest mortality, proportionally to the numbers of old people living. Tuberculisation is rare, but tuberculous pneumonia is not; the most common form is cavernous phthisis, and especially the fibrous kind, which, after a long silence, may be revealed by accidents or unexpected complications, which are frequently mortal.

SIXTH PART.

Hygiene.

XXXVII.

EXTERIOR PRESERVATION.

Phthisis, in future to be classed amongst virulent maladies, must naturally be subject to the laws of contagion; but these laws vary, so to speak, for each contagious malady. Infallibly inoculable, whether by the aid of tubercular tissues, whatever their sources, or by the sputa of phthisical patients, phthisis presents as to inoculability a perfect analogy with small-pox and syphilis. The difference is only in the resistance of living species or animals submitted to experiment. Whilst syphilis and small-pox are so easily transmitted to man, and with such difficulty to animals, we see in phthisis exactly the reverse. At present, in truth, tuberculous matter, supposing that it penetrates accidentally by the integuments or superficial mucous membranes, has never given rise in man to any local contamination, nor, for a stronger reason, to any general infection. Tuberculous contagion is exerted in two ways: (1) by the digestive tract through the agency of food; (2) by the respiratory organs, that is, by the atmosphere.

§ 120. *Alimentary Preservation.*

The tubercular virus may penetrate into the organism by the aid of food; the muscular tissue, and especially milk, coming from animals of the bovine race attacked with tubercular disease, should be held in grave suspicion.

Bouley, in his marvellous lessons on the living nature of contagion, has demonstrated that, according to his researches, and those of Lynt and John, phthisis is very frequent amongst oxen and cows; it attacks pigs and fowls less, and spares sheep. The bovine race in certain countries is attacked in the proportion of two per cent. The abattoirs show us this proportion in an evident manner, particularly in hot countries, much less in cold, as Sweden and Norway, although there the beasts are *feeble and rachitic.* This, by way of parenthesis, would lead us to infer that amongst men the feeble or the enfeebled do not pay a greater tribute than the others. The age of the affected animals is always about the same. We find phthisis much more rarely in very young animals than in those aged from six months to three years. Here, again, is a certain analogy with what passes in the human species; new-born children up to one or two years are rarely tubercular.

Lastly, here is a dominant fact with regard to the origin of human phthisis. There exists, according to Lynt, a true parallel between bovine and human phthisis; the curves of double mortality are the same for different districts of the Duchy of Baden. We can only explain this coincidence by the use of tubercular flesh or infected milk.

I. *Infected meat.*—Grave consequences arise with regard to the sanitary inspection of animals. Perhaps meat should be submitted to a rigorous examination, and that proscribed which comes from cattle attacked with caseiform indurations of lungs or glands.

With regard to this point in human preservation, I rely on the authority and sagacity of the celebrated veterinarian. The danger is so much the more considerable, as cooking the muscular tissue at 62° C. in no way destroys its virulent properties (Toussaint).

For stronger reasons, the use of raw meat, or what we call *bleeding*, that is, raw at the centre, should be prohibited; it is for this motive that Bouley recommends mutton, sheep being very refractory to tuberculosis.

II. *Bacilliferous milk.*—We have already discussed this question, § 37. If the milk comes from a cow attacked with general tuberculosis and with the udder itself affected, the transmission of bacilli is not doubtful. Happily, coction at 100° C., as recommended by the Congress of Baden, 1882, is sufficient to deprive the milk of its dangerous qualities. This is, then, an indispensable precaution to take in presence of the slightest suspicion of the pathological origin of the milk.

§ 121. *Atmospheric Preservation.*

I. *Air vitiated by invalids.* — Contagion for man is exerted especially by the atmosphere, but only by *a special* atmosphere. We believe in reality that it is not by the surrounding air which we breathe inside and outside our houses. If respired air contained these parasites developed, formed, and ready to invade us without ceasing; if our atmosphere was in the power of these deleterious beings, as in marshy countries where it is filled with organisms of fever, the human race would long since have disappeared from phthisis. Happily, contagion by air, that is, penetration of the tubercular virus into the respiratory organs of man, is surrounded with all sorts of difficulties and numerous conditions, without which the transmission is impossible.

First of all, the air must be contaminated by the phthi-

sical patient, that is, by air which he has breathed, or especially from *debris* of the products of expectoration. Dust from dried sputa, spread through our habitations and mixed with the dust from clothes or carpets, is a true cause of vitiation of the air; here is a grave danger. It seems from these data that nothing should be more easy than to destroy the infecting agent by operating on the mucosities expelled from the bronchi into the vessel or linen used by the patient. To this end all kinds of disinfecting agents have been used; finally, they are rendered harmless by concentrated alcohol or carbolic acid, which, in the proportion of five per cent., will neutralize the virulence of the *crachats*. It has been proposed also to submit these virulent substances to the action of fire. An elementary precaution which should be always observed is not to allow expectorated materials to remain in the chamber of the patient.

II. *Atmosphere of cities and overcrowded bodies of men.* —If one can follow, so to speak, contagion, when the air is impregnated with the dust of dried *crachats*, it is no longer the same for that mysterious contagion which is unceasingly exercised around us by patients unconscious of their state, and on individuals who are not alarmed by the presence of these dangerous neighbours. We know that cities are permanent centres for the transmission of the microphyte, and that the mortality is infinitely more considerable than in localities where inhabitants have but rare contact with the world which is called *civilized*.

How shall we deal with immigration into these great centres? Agriculture is abandoned; life in the fields a myth, and crowding together the rule. Under this name, overcrowding, we must include especially the collection of children in schools and colleges, women in workshops, men in barracks, prisons, ships, etc. Wherever life is in common in a confined space, the microphyte is to be feared. A single

tubercular sufferer, even in the latent or initial stage, is sufficient to infect the air by expectoration. Insufficient aeration is much less to be feared with regard to phthisis than a vitiated virulent atmosphere. What shall we do with regard to these necessities of existence? Ventilation only affords a momentary purification; cleanliness, especially with regard to clothing which may be impregnated with bacilliferous dust, is a precaution really more important. One sees, alas! how much sanitary measures are restrained in application, and without results. The dispersion of individuals and their isolation is not within the range of practice.

§ 122. *Individual Preservation.*

Matrimonial contagion.—The most frequent and best proven means of contagion is through marriage. In all times, in all countries, numerous examples have been cited as to the transmission of the malady from husband to wife, and perhaps more often in an inverse manner; inquiries made as to contagion are unanimous as to the danger of cohabitation. An affected woman has contaminated several husbands successively, which supposes a survival of some years, either in the insidious latent period, at the start impossible to be recognized by auscultation, or of a more advanced stage, easy to be seen, manifest to all, but yet compatible with a certain degree of strength, good condition, and an appearance of health. In one or the other condition there exists a microphytic infection which is easily to be found by a microscopic examination of the *crachats;* it is the microphyte which is transmitted. Generally contagion takes place through air expired from bronchi filled with bacillary secretion, or rather, by air which has passed over expelled sputa, which are more or less dried. One can easily conceive the dangers of a too intimate neighbourhood, and of cohabitation which multiplies to infinity the absorption

of virulent products. The practical conclusion is easy to foresee; there is danger of exhaustion for the phthisic, and danger of contamination for the healthy person, whether husband or wife.

Contagion in general.—Contagion in general is more rare, and as it is exercised especially by the respiratory tract, it is important to purify the air in an absolute manner, that is, to free it from microphytes and vegeto-animal dust. To attain this end we must disinfect the products of expectoration in a permanent manner, either by absolute alcohol or carbolic acid, and prevent their being kept in vessels placed at a greater or less distance from the bed, also remove soiled linen, and completely ventilate the chamber as soon as the patient has left it. We must avoid the collection of dust on the furniture, hangings, etc. Other precautions would be useless to the assistants, and would cause the patient grave moral injury. As to the practitioner, although he may be imbued with contagionist doctrines, his first duty is to be silent about them, his first care to make the people take the indicated precautions on the ground of necessary cleanliness.

§ 123. *Preservation of Families.*

Heredity, which for the attentive observer is undoubted, should not, however, be regarded as the sole cause of the fatality which weighs on certain families. We must certainly not consider as hereditary the cases of contagion which result from life in common with a direct or collateral relation, who is attacked by the malady; with such unfavourable conditions transmission may up to a certain point be avoided, and the precautions indicated with regard to contagion in general will contribute powerfully to preserve those who seem destined for an unhappy fate. There is the strongest reason, if one can, to divide the family

and bring up the children away from the parental house. Abstraction being made as to this family contagion, there remains always transmission, whether direct or, as it is said, by predisposition. It is not surprising that the virus propagated by the father or mother may rest in a latent state in very young children, and that in general it is not developed until the age of five up to ten years; then in the form of scrofula, later in the form of pulmonary phthisis. We must remember that syphilis, which is manifestly hereditary, and, so to speak, inoculated to the child by one or other of the parents, may equally delay in unmasking itself, as Professor Fournier has demonstrated. The bacillus may pass by the blood of the mother, through the placenta into the blood of the child, and remain in its body, which begins by being refractory, as are certain animal races. Can we prolong this stage of resistance, not to the invasion of the bacillus, which has already taken place, but to the action of the parasite, to limit and, so to speak, as Baumgarten expresses it, encyst the bacillus in the organ primitively attacked? If there are prophylactic means, they ought, at the time of the development of the child, to contribute in rendering sterile the soil which may be capable of developing the microphyte. We have a small number of available resources. (1) Gymnastic exercises, particularly those which develop respiratory power and capacity. (2) Hydropathic means, as sea baths. (3) Food, which must be regulated (see § 37 and § 38) so that there is a predominance of fatty materials, and especially avoiding any excess in the quantity of potash salts.

§ 124. *Preservation of the Predisposed.*

The difficulty of admitting either a direct transmission of tubercular mischief, or, as we actually believe, of the

bacillus tuberculosis, has given rise at an ancient date to the idea of the transmission of a simple predisposition or predestined constitution. These are words contrived to disguise our ignorance on the subject of heredity in general, and of the family propagation of phthisis in particular; but in avowing our doubts as to the bacillary impregnation of the fœtus, we cannot resist showing the danger of this denomination, so vague and so simple in appearance—*predisposition.*

How shall we treat a physiological entity which is neither definite nor recognizable, and by what measures?

Physiological misery.—They say that the future tubercular patient is a wretched being, whose whole organism, muscular and osseous systems, particularly in the chest, have been arrested in their development. Also, that nutrition is defective, imperfect; that leanness and anæmia are its signs in consequence; and lastly, that the malady attacks especially those who are badly nourished, badly lodged, and who live, as cruelly expressed, on privations. All this constitutes physiological misery, atrophy, which is said to be the prelude of phthisis, its necessary preliminary condition. But Schlokow attacks the theory of phthisogenic misery with certain facts (Soc. Méd. de Berlin, 1883).

In analyzing the phenomena one perceives quickly enough that there is an error, a dangerous interpretation; it is sufficient to know that amongst those families who pay the sad tribute to fatal heritage, there are individuals who have good looks, the most flourishing health, luxurious conditions, and irreproachable hygiene, but who surprise the physician in the midst of complete security, whilst a brother or a sister, who seems by miserable constitution exposed to all the dangers, all the eventualities of the malady, resists and remains impassive to all attacks from the family evil. This is the error, the interpretation is yet more grave; for

thus the strong are neglected, whilst the weak are attended to with minute care, so as to effeminate them under the pretext of hindering colds; or too active means, which are intended to inure them, are used.

§ 125. *Hygiene for the Feeble.*

Vicious conformation of chest.—In tubercular or even healthy families we often meet individuals who by their small size, their atrophic aspect, and especially by the imperfect development of the skeleton, seem the predestined victims of phthisis. We know already that these feeble natures resist hurtful influences perfectly, particularly parasitic infection, and that they are not more subject than those of strong constitution to contract contagious maladies. For these, then, there are no preventive measures to apply, all our attention should be directed to the badly formed chests, especially with exaggerated retraction of the upper part, and very feeble muscular system, which is incapable of producing large ampliation of thoracic cavity. Here we find a favourable condition, if not for the reception of the parasite, at least for its multiplication. In truth, with such conditions the bacillus finds in the lung its true field of culture, a sort of shut sac inaccessible to vivifying air, and the seat of immovable mucous secretions. We know that when oxygen easily reaches microphytic colonies, it breaks them up or hinders multiplication; and, on the contrary, they multiply in albuminous stagnant liquids. We must, then, favour the absorption of oxygen, and hinder the stagnation of mucous secretions in the bronchi; the problem is solved by respiratory gymnastics. Clark had reason to say that with the rich the true misery which predisposed to tuberculosis was a sedentary life.

Irritability of mucous membranes.—The question of predestination seems to be altogether in the disposition *to*

take cold, that is, a special native irritability of the bronchi, which under the least influence of cold, or under the impression of the least dust, become the seat of acute or chronic inflammation either continued or at intervals. The kind of reasoning which traces phthisis from this bronchitis is as follows. Tubercle, they say, is an inflammatory product of the lung; bronchitis is inflammation of bronchial mucous membrane. From the bronchi to the lung is but a step; inflammation is propagated by contiguity and continuity; tubercle is the effect. After the example of Laënnec, who saw all, comprehended all with his penetrating genius, we protest energetically against this dangerous opinion, which in France still reigns everywhere, in society as in the medical world. The latter, because it believes bronchitis may be transformed into tubercle, orders infinite precautions against cold and heat, against wind and dust, dryness and humidity; inflicts cruelly on the invalids preventive blisters, preservative fire-points for fear of repetition of malady, prophylactic cautery against perpetuity, and even a choice of defensive setons to drive off existing inflammation or conjure away the future phlegmasy. Here, however, is the *modern* reasoning, which places bronchitis amongst the causes of wounds of respiratory mucous membrane; following inflammation comes ulceration, which opens the door to the bacillus, that without this would be lost on the surface of the mucous membrane of the bronchi. Thus the more one has suffered from bronchitis the more we are open to the microphyte. We must, then, try to cure this bronchitis at the commencement, these chronic catarrhs and repeated inflammations; we must, at all costs, hinder them from becoming ulcerative, which is the more easy as the erosions are unknown. It is necessary to prevent relapse, and arrest the predisposition. To do this, they (French practitioners) place their patients in warm rooms, inac-

cessible to air; and, if they are obliged to leave these incubators and face the air, the mouth is to be covered in English fashion with a respirator containing cotton-wool, which protects from cold, and filters the air, stopping the bacillus on its way. As there does not exist on the surface of the globe a single individual who has not had more or less cold and bronchitis, the people and the great world should unite, remain at home, so as not to take cold again, and isolate themselves so as to avoid contact with atmospheric bacilli. These are coercionary measures which the new form of their doctrine demands. Laënnec would have laughed at them as at the partisans of the ancient phlogistic doctrine. He said, in fact, "People do not become tubercular as a consequence of neglected colds, they are so already. The neglected cold is, then, a sequence, and not the cause of the malady."

It is often difficult to know if bronchitis is simple or tubercular, on account of the negative and uncertain results from auscultation at the beginning of the malady; there remains the resource of the microscopic examination of sputa. Whatever may be said of it, the microphytic diagnosis is so precious that it precedes all other methods of investigation by months and often years. If you do not find the bacillus in these *neglected colds*, it is because you have to deal with common catarrh, which may be treated with a pectoral mixture and patience.

TRANSLATOR'S NOTES.

After this vigorous and ironical attack on the doctrines of a modern French school, we note with some surprise the omission of certain important facts. The influence of a dry atmosphere, as in the northern regions, is proverbial.

Welch says, "During winter, clothes put out wet lose their moisture more rapidly than in an English sun. Such is the evaporation from the surface of the ice, that a stone originally attached beneath finds its way to the top. Moisture in the solid state is abstracted from every material possessing it. *The effect of this on all organic particles is palpable, they desiccate and become innocuous.* In this probably lies the apparent anomaly in Spitzbergen, Iceland, and Arctic zone, of great overcrowding and vitiation of air, with almost complete absence of all kinds of pulmonary disease."

Morgan (Brit. and For. Medico-Chirurgical Review, vol. xxvi., 1860), in an article on "the non-prevalence of phthisis in the Hebrides and along the north-western coast of Scotland," draws attention to the influence exerted by a peculiar construction of dwelling-houses. He says, "The inmates of these huts enjoy, if not an altogether perfect, at least a very efficient system of ventilation. The peat smoke is seen escaping not only at the smoke-hole and the door, but through innumerable crevices in the walls of the house, showing they are pervious." The introduction of "chimneyed cottages" was shown to have been a factor in the subsequent increase of the disease. It appears certain that M. Sée under-estimates the importance of hygienic defects such as want of proper ventilation in dwelling-houses.

Buchanan (Ninth and Tenth Reports of the Medical Officer to the Privy Council, 1866 and 1867) shows clearly that in a number of English towns, which have been properly drained, and in which the ground has been rendered much drier, the mortality from phthisis has also been much reduced.

SEVENTH PART.

Therapeutics.

XXXVIII.

CLIMATIC THERAPEUTICS.

CLIMATE is a medicament composed of the common elements—oxygen, temperature, light, movements of air, barometric condition. · All these things constitute, all of them complicate, a climate which for us henceforth is but a physico-chemical mixture, to be studied with regard to physiological effects and especially curative action. I add a second kind of element, which will in future serve the educated clinician, who is careful of his scientific dignity—that is the vital composition, if we may so express it, of the atmosphere he is about to prescribe. Is it pure, that is to say, exempt from microphytes in general? Is it hostile to the life and the multiplication of bacilli, which have already invaded the bodies of those whose care is confided to us? These are the questions which start up in the therapeutics, which we may call climatic.

Therefore, taking as a starting-point, the double question as to the vitality and physico-chemical state of the air, I am able to divide these climates into three categories, of which the two first are the most important in the therapeutics of phthisis.

1. Warm maritime climates.
2. Climates of altitudes which are necessarily cold.
3. Low-lying, warm, non-maritime countries.

§ 126. *Climates of Altitudes.*

If we place ourselves in the only true point of view, we shall recognize the disappearance of the profound differences which in recent times have existed between the partisans of warm countries and those who believe in altitudes. The conditions of the development of tubercular bacilli may be diminished, or even avoided, in both localities which are so antagonistic from a climatic point of view. In cases of amelioration or cure, we thus, without doubt, transform phthisis into a local circumscribed tuberculosis, so to speak, *enclosed.*

(The English reader will find information as to Davos Platz, Saint Moritz, and other stations in the Engadine, by referring to various monographs on the subject.)

Mountain stations. — Miquel and Freudenstein have proved that, at the height of 1800 mètres above the sea-level, there are no longer any parasites in the air, and it is to this quality that we must attribute the prophylactic effect. The general characters of mountain climates may be thus condensed: (1) The air of mountains has a less pressure; it is more fresh, although the sunshine is warmer than in the plains; it is drier in spite of abundant effluvia; and it is in summer more disturbed by winds than during winter in elevated and protected snow-valleys. (2) The air is remarkably pure, relatively, to all miasms, and all inorganic or organic products. (3) Lastly, ozone is present in marked quantity.

Effects on the healthy and invalids.—1. At the beginning of residence at a mountain station the heart presents a

marked activity as to energy and number of beats; later there is a return to the normal state, with conservation of contractile force and power of aspiration.

2. The respirations are more frequent at the start; the thorax dilates one or two centimetres (Bauer). At the end of a certain time the respirations become normal, but probably preserve more energy. Nevertheless, the lungs are distended with blood, and *perhaps* there is danger of congestion (Weber).

3. The consumption of oxygen is reduced to a minimum; 348 grammes less daily than at the sea-level (Jourdanet).

4. The elimination of water, and especially carbonic acid, by the lungs is manifestly increased. Oxidations are doubled. Marcet has noted at 4000 mètres 15 per cent. more CO_2 than near Lake Geneva, at 380 mètres. This is one of the most remarkable physiological effects.

5. The appetite is increased, and in consequence of the nutrition of organs the nervo-muscular power, and perhaps the oxidations are more marked. We may say in general that mountain climates are exciting. They stimulate most of the functions, exercise a strengthening influence, but they require a certain resistance of constitution.

Therapeutic effects.—This purity of the air so much governs the question of altitudes, that they are used in treating hereditary or acquired predispositions to tuberculosis, that is, preservation from bacillary infection, rather than in the treatment of distinct phthisis. Weber has shown that the therapeutic data are scanty and uncertain. The diminution of respired oxygen, the low temperature, and the presence of ozone, have all been evoked to explain the beneficial effects recorded by different observers.

§ 127. *Warm Maritime Stations.*

(The reader is referred to different classical authorities on climate for the necessary local particulars given in the French edition.)

General properties of maritime climates.—(*a*) Despite the humidity which always exists, the air of the sea presents a certain uniformity of temperature in summer and winter, and even from night to day. If, during the day, there is sudden lowering of temperature, the reason is often to be found in local dispositions. They are less found at Mentone than on the rest of the Riviera.

(*b*) Atmospheric pressure is very high, but the oscillations are considerable and regular.

(*c*) The air is generally, up to a certain point, disturbed by winds of local origin (land and sea breezes), but further there may be wind of foreign source.

(*d*) Light is intense, although clouded by atmospheric mists; and light, according to the researches of Downes and Blunt, is the enemy of bacteria and spores, so much so that it hinders their development.

(*e*) Under the influence of solar light, of evaporation, and movements of air, ozone is always present in considerable quantity, which perhaps explains the vivifying influence of sea air on the general constitution.

(*f*) The air is free from dust, whether mineral or organic (excepting the organisms of malaria). Although under the influence of the wind, a great number of localities contain saline substances, bromine and iodine, in the atmosphere. We know that these bromo-iodated saline matters are not found at a greater distance than 200 or 300 mètres from the sea.

Physiological effects.—Beneke, who has studied the physiological effects of sea air at Nordseebad, has remarked

that the body is more easily chilled than in sheltered situations, and especially more so than in the mountains, which necessitates greater covering with an equal temperature. He has noted also activity of oxidation, as shown by excess in production of urea and sulphuric acid, diminution of phosphoric and uric acids, augmentation of body weight. To these effects we must add a slight slackening in respiration and pulse, compared with what passes in regions situated higher than 100 to 300 mètres. This circumstance appears due to excess of pressure and humidity. We may, in a word, consider this maritime climate as calming and at the same time strengthening.

Therapeutic effects. — Maritime residence, abstraction being made as to sea-baths, constitutes a precious means of preservation from scrofula, and even phthisis, which often follows it. The beneficial effects, noted by Williams, father and son, and by Weber, in the treatment of phthisis, appear due to three causes: (1) The high barometric pressure, which, by accumulation of air and oxygen in the lungs, seems opposed to the existence of the bacillus; (2) the great quantity of ozone which produces the same effect; (3) the bromo-iodated substances which have an incontestable effect, inasmuch as they are antiseptics; they even constitute the principal beneficial factor in sea air, and explain its superiority to the neighbouring atmosphere of the continent.

Comparison between these two climates.—The common conditions are purity of air, more or less complete absence of dust, and consequently of micro-organisms in the atmosphere, great light, and very marked ozonisation. The specialty bears on the presence in marine air of bromine and iodine, which act as true antiseptics. In altitudes it is the low temperature which acts in a particular manner. The real difficulty is to reconcile the benefits of the two climates with the differences which separate them with

regard to barometric pressure. How is it that, on the one hand, *excess* of pressure at the seaside, and, on the other hand, *rarefaction* of the air, with diminution in pressure, on the mountains, are both equally advantageous to the patients? The contradiction is only apparent. Predominance of oxygenated air is manifestly contrary to the biological properties of the bacillus. Again, respiration in a *rarefied* atmosphere, with marked diminution of pressure, singularly facilitates complete aëration of the lung, and consequently hinders the bacillus from stagnating, that is, from multiplying. It seems to me that these interpretations, which confirm all clinical data, have nothing hypothetical in them. They rest on the study of the laws of parasitic development.

§ 128. *Low-lying, Warm, Non-maritime Countries.*

Certain of these enjoy a great reputation. Pau has often been compared to Rome and Pisa, but with incontestable superiority due to purity of air. *Egypt:* Cairo, Nubia, and the banks of the Nile are all famous resorts at certain seasons of the year, usually in winter. The influence of forests is marked in obtaining uniform temperature, although with considerable humidity. When the trees are of a resinous nature the influence of ozonised terebenthinate products comes into play, and may perhaps be useful to the patients.

XXXIX.

CURE AND THERAPEUTIC MEASURES.

§ 131. *General Idea of Anti-virulent Treatment.*

One single idea dominates medicine, and especially therapeutics, to-day; it is the influence of the parasitic kingdom on the human organism, and the conception of medicinal

means destined to oppose the life and reproduction of these morbigenic microphytes. But already dissensions have begun, and are increasing, whilst eminent physicians query, and even proclaim the uselessness of the attempt, contenting themselves with the old dogma of contagion, because they think to recognize the mischief without being able to combat the contagion (we can neither treat nor cure a philosophic abstraction); because the discovery of the bacillus seems to them, good at most for making or completing a diagnosis; because, in a word, the doctrine will be useless, as the application to internal medicine must be without results. On the other hand, we see in the opposing camp the most ardent partisans of the microphytic theory advancing rapidly, perhaps even rashly, towards the research of necrobiosis of the bacillus, encouraged as they are by the marvellous examples which have been quoted in surgery and obstetrics, by the striking effects which have been obtained in the treatment of wounds and puerperality. People cast stones at us *medical* practitioners because up to the present we have not been so happy:—*væ victis;* but we shall not be the vanquished at the end, and the new path on which clinical medicine might and ought resolutely to enter will not be sterile. We are not daunted by the mistakes of a first beginning, and in reality the difficulties are much more considerable than in surgical or obstetrical science, where necrophytic treatment finds the parasite, so to speak, to hand. In medicine the parasite, instead of resting on the surface as of a wound, has penetrated profoundly into the tissues, bronchial mucous membrane, or into the lung. You will not attack it with fumigations, nor even with intra-pulmonary injections of dangerous anti-virulent substances. The enemy is in possession, and not about to enter the economy. We must attack it indirectly, and to do so must impregnate

the affected organ with the medicine, without exposing the organism to danger. The medicament, if it cannot reach the bacillus, must strike it indirectly in vital conditions, in its means of existence. This is the secret of certain useful medicinal actions; it is the source of curative indications.

§ 132. *General Indications of Treatment.*

The general indications of treatment are of diverse orders.

1. To hinder the parasites from living or multiplying, or freeing the limits of pulmonary regions; in other words, to hinder fructification and diffusion of microphytes with anti-parasitic means.

2. As the microphytes already exist in more or less considerable number when we are called upon to treat the patient, it will not be enough to prescribe an anti-microphytic, whatever its antiseptic power. It is necessary that this destructive agent of the parasite be reconstituting for the individual. What damages the offending beings must benefit our organism; in other terms, the anti-virulent must be a trophic agent.

3. If this anti-virulent agent, which we may call necrophytic, does not possess trophic properties, it should at least act on the respiratory organs, that is, on their constituent elements or on their functions; in a word, it must be at the same time a modifier of the mucous membrane and broncho-pulmonary innervation, that is a *respiratory medicine.*

4. Any treatment which does not modify general nutrition or the local functions of the lung can but constitute an imperfect resource, but one of which the utility cannot always be contested; indeed, we find in medicinal foods powerful therapeutic auxiliaries. Favourable effects on the respiratory organs are also to be obtained from certain medicines which modify the texture and secretion

of the bronchi, either by indirect way or by artificial atmospheres. All the rest concerns but the symptoms of ordinary phthisis or its various forms. Let us commence with the anti-parasitics or necrophytics.

§ 133. *Necrophytic Medicines.*

Demonstration of their necrophytic power.—Chemical agents are the only means of attacking the parasites in the living body. Their effects do not become manifest, that is deleterious to the microphytes, but with large doses especially as compared with those necessary to protect us from the same influences. We may test their anti-virulent or disinfectant power by operating on sterilized culture-liquids, as well as gelatinous broth, or extract of meat, or serum rendered sterile by heat, filtration, or hermetic sealing, from all common infecting agents. Koch, in trying different substances, calculates how many days are necessary to annihilate the bacillus anthracis.

Buchholz cultivates the parasites and determines the quantity of disinfectant necessary to hinder their development.

Miquel has prepared a table of antiseptic substances according to the gradually increasing quantity which is necessary to preserve a litre of *bouillon* from putrefaction. Despite the divergence of these proceedings, the different experimentalists have arrived at nearly the same results. The following is an enumeration of the substances which are applicable to man.

1. *Salts of mercury. Corrosive sublimate. Biniodide.*—Koch says that solution of perchloride 1 to 300,000 is sufficient to kill bacilli in a day. Buchholz speaks 1 in 20,000 for hindering the development of the parasites. Miquel says that five centigrammes per litre of *bouillon* will arrest putrefaction. Further, in his recent experiences he

recognizes a yet more powerful influence in the biniodide of mercury, since 25 milligrammes serve the same purpose. This is not surprising, for the action of this salt comprises that of the two most powerful necrophytics, mercury and iodine.

Subcutaneous injections of perchloride as a necrophytic measure.—In general, the mercury produces salivation, diarrhœa, anæmia, prostration, and amongst those who can tolerate the remedy the improvement of symptoms has no duration. Hiller has treated forty-four patients by this method.

Perchloride by direct injection into the lung.—Hiller has also tried the parenchymatous injection, the dose being two centigrammes in solution. In all three cases he was obliged to suppress the treatment. This want of success in treatment of phthisis by mercurials is perhaps surprising, if we take into account the marvellous results obtained from the perchloride of mercury in surgery, midwifery, and lately in ophthalmic practice as a preventive and curative agent in the ophthalmia of new-born children.

2. *Oxygenated water. Permanganate of potash.*—Both these powerful antiseptics are very difficult to manage, and at present scarcely within the range of practice.

3. *Iodine. Iodoform. Iodides.*—These will be described in a special chapter. Miquel states that from twenty-five to sixty centigrammes of iodoform are necessary to preserve a litre of bacilliferous *bouillon*, whilst 140 grammes of potassium iodide must be used for the same effect.

4. *Arsenic. Arsenious acid. Arsenites.*—The antiseptic power of arsenious acid and the arsenites is much less than that of iodine and iodoform. Miquel had to use from six to nine grammes of these substances per litre to obtain real preservation. Their utility in the treatment of phthisis is not less incontestable, and depends on other properties.

5. *Aromatic acids.*—The aromatic acids, salicylic acid, and the salicylates act more feebly than iodine; their salts are less active than the acids themselves. Amongst these aromatic compounds, we must mention especially salicylic acid, which acts when from one to ten grammes per litre are used (Miquel says ten times the quantity of salicylate is required). Benzoic acid acts in proportion of two grammes per litre; according to Buchholz, benzoate of soda is efficacious at $\frac{1}{2000}$. Carbolic acid does not commence its preservative action until from three to twenty grammes per litre of culture *bouillon* are used.

6. *Alcohols.*—We may mention amylic alcohol, which is much less antiseptic; fourteen grammes are necessary to act on a litre of sterilized *bouillon;* glycerine is still less active.

7. *Ozone. Turpentine. Creosote.* — Ozone has an undoubted action; the other two substances owe their antiputrid action to its influence.

Differences of necrophytic action on different microphytes. —The chemical substances which serve to nourish one form may be poisonous to another; thus solution of potassium arseniate enables one species to vegetate, but becomes poison to a parasite of superior order. In solutions of quinine at $\frac{1}{20}$, colonies of moulds and microphytes are developed, whilst weaker solutions are sufficient to disinfect liquids containing other micro-organisms. A question suggests itself on this subject: Are morbigenic species fixed, or may they combine and create varieties? Certainly, new diseases have appeared and others are extinct; the plagues of the Middle Ages have disappeared; diphtheria, recurrent fever, are of recent date; but in reality this fatal family of microphytes dates from afar, and remains immovable and immutable; there are true species which are reproduced indefinitely by culture whose morphology may be similar,

but whose vital morbigenic action differs totally. That is why all antiseptics do not enjoy specific necrophytic properties.

§ 134. *Inhaled Necrophytic Medicines.*

As soon as the bacillus was discovered, people began to pursue it directly with the aid of volatile reputed antiseptic substances, and which were launched on the *locus minoris resistentiæ.*

Let us now see the methods of attack. The medicines may be in the form of solid particles, in the liquid state, or in the form of gas or vapour.

Solid substances, when they are soluble, act as liquids in the respiratory organs, dissolving in the laryngo-bronchial mucus; when they are insoluble they are in part expectorated, and in part fixed by broncho-alveolar epithelia. We know in reality that powdery substances, as charcoal (Traube), or metals (Zenker), or vegetable products like cotton, or, lastly, animal dust as wool, all penetrate profoundly into the epithelium of alveoli, into the interstitial tissue of the lungs, into the lymphatics, and from there into pleuro-pulmonary parenchyma, where they form grave lesions, chronic caseiform ulcerative inflammations, absolutely analogous to those of tuberculosis. It is very difficult to introduce powdery insoluble bodies into the respiratory tract. Most frequently they stop before arriving at the place of destination, that is at the upper extremity of the bronchi. It is necessary in all cases that they should be drawn in by a current of air.

Inhalation of vapours.—Different anti-infectious agents have been tried under the form of inhalation, by Fräntzel in conjunction with Koch and Gaffky.

Experiments have been made with those medicines which *in vitro* are opposed to the ulterior development or culture

of the bacillus, and consequently to the multiplication as well as the functions of the parasites. People have chosen naturally those which can be mixed in the air in the form of vapour, and penetrate the respiratory apparatus, from which the patient imbibes the medicinal air. This apparatus consists of a galvanized iron box, easy to aërate, and lined with a layer of paper containing the medicine. The patients breathe this for as long as eleven or twelve hours daily.

Choice of vapours.—The vapours of mercury, and carbolic acid have been tried on twenty-seven patients without the least effect. Creosote itself was useless in this form, whilst it offered real advantages as an internal medicine.

Inhalation of gaseous medicaments.—Other methods have been employed by Hiller, amongst others the inhalation of gas, or of very finely divided vapours. In the coarser form no vapours penetrate into the pulmonary parenchyma; the mucous membrane of the pharynx, larynx, trachea, and of the large bronchi, stops them on the way, or causes their expulsion by coughing. Also there are few antiseptic gases capable of being breathed without danger; chlorine, bromine, sulphurous acid, sulphuretted hydrogen, irritate the respiratory mucous membrane, or become toxic, if they are not mixed with a great quantity of air, and in the latter case action on the bacilli is impossible; it is extraordinary if they reach the *walls* of cavities.

Therapeutic results.—Hiller employed separately or successively on the same patient, (1) corrosive sublimate, (2) iodoform, (3) bromine, (4) ethyl alcohol, (5) methyl alcohol, (6) the gas sulphuretted hydrogen, (7) arsenious acid, (8) boric acid, (9) salicylate of soda. The effects were absolutely negative; they did not succeed in arresting the tubercular process, nor in making the bacilli disappear from the sputa. These experiments were performed on eighty-one patients at the *Clinique* of Leyden.

Respiration of natural gases, absorption by pulmonary vessels.—The gases properly so called are introduced without any preparation, as air, into the respiratory tracts; there they are absorbed, and this absorption is made less by the respiratory mucous membrane than by the immense capillary network which covers the lungs. Oxygen, pure or active, nitrogen, carbonic acid, and sulphuretted hydrogen, penetrate easily by respiration. We shall especially study oxygen and sulphuretted hydrogen.

XL.

PLAN OF THERAPEUTIC STUDIES.

First empiricism, and afterwards experience, have put precisely in the first rank of curative means for tuberculosis, the best-proven necrophytic agents, which enjoy at the same time either the power of lessening oxidations, or the faculty of modifying respiratory functions or organs. Regarding medicaments from these points of view, we shall find three entirely distinct series.

First Series. Necrophytic, Trophic and Respiratory.

1. *Iodine, iodoform, alkaline iodides.*—All these preparations enjoy the triple privilege of being anti-parasitics, as well as modifiers of oxidations and respiration.

2. *Arsenic and its compounds.*—Arsenic, without being an active antiseptic, constitutes a means of strengthening the organism indirectly, and hardening it against the development of bacilli; in every way it is a most evident means of saving, and at the same time a powerful auxiliary of respiration.

Second Series. Trophics or Lesseners of Expenditure.

1. *Fatty medicines and foods.*

(*a*) Neutral fats.

(*b*) Cod-liver oil.

(*c*) Glycerine.

(*d*) Milk; milk treatment.

2. *Properly called lesseners of expenditure.*

(*a*) Alcohol and its derivations.

(*b*) Gelatine.

Third Series. Oxydizers and Ozonizers. Aromatic Acids.

1. Oxygen.

2. *Ozone.*—Oxygen constitutes one of the most efficacious methods for opposing the development of bacilli; under the form of oxygen-ozone it is still more active, but difficult to manage.

3. *Benzoic acid and benzoates.*—We know already their antiseptic power, and their favourable action on mucous membranes cannot be contested.

4. Creosote.

5. The turpentines have ozonizing and especially secretory properties.

6. *Sulphur, sulphides, hyposulphites.* These substances are reputed antiseptics, and appear to enjoy a marked action on the secretions.

Fourth Series. Inorganic Foods, Alkalies, Phosphorus, Hypophosphites.

FIRST SERIES.

NECROPHYTIC, TROPHIC, RESPIRATORY.

We have to study with regard to the treatment of tuberculosis: (1) iodine, (2) alkaline iodides, (3) iodoform.

XLI.

THERAPEUTICS OF IODINE.

The curative effects are due to different, perfectly defined properties which we shall enumerate in the following order:—

1. *Antiseptic properties of iodine.* — The first most clearly established property of all relates to its antiseptic or necrophytic power, which corresponds exactly with what is called its specific action.

2. *Secretory action.*—Absorbed iodine is eliminated by the mucous membranes, and in passing through these tissues modifies them profoundly as well as their secretions; this action is precious in the treatment of diseases of the respiratory organs.

3. *Lessened expenditure and organic changes.*—It is especially a medicine of economy for the general nutrition, at the same time a profound modifier of physiological tissues, and especially certain neoplasms.

4. *Respiratory action, and that against dyspnœa.*—We may consider it further the most powerful of the medicines that I call respiratory, but whose mode of action on the respiration has passed totally unperceived.

5. *Vascular and nervo-vascular changes.*—It further

modifies local circulation, either directly, or by the intermediate influence of vaso-motor system.

§ 135. *Iodine. Preparations, Doses, Absorption, Elimination.*

§ 136. *Ingested Iodine. Iodide of Ethyl by Respiration. Iodine applied on the Skin.*

Ingested iodine. Physiological effects of therapeutic doses. Local effects.—Iodine applied to a mucous membrane acts as a caustic, although with much less energy than the caustics; that is why Majendie justly said we should disregard its irritant action. In truth, iodine mixed with saliva acts but moderately on the stomach; that is why we may with impunity carry the dose to four, six, or even ten grammes daily. The local action need not detain us.

Toxic doses.—Certainly iodine absorbed by mucous membranes, wherever they may be, may determine accidents. We remember the disastrous effects produced on great absorbing surfaces, as of the pleura or cysts. Thirty grammes of tincture of iodine diluted and injected into a cyst have produced death (Hermann). In the famous case of the surgeon Rose, who injected 150 grammes of tincture, with 3·6 grammes of potassium iodide, the whole diluted with 150 grammes of water, toxic accidents were manifested at the end of six hours and continued till death, which took place on the tenth day.

Effects of return to the stomach. Vomiting.—In all cases of iodic intoxication vomiting is seen with evident lowering of temperature, and a sort of *contraction* of arteries.

Elimination of iodine by the stomach.—The vomited matter *always* contains free iodine, and in Rose's case the vomit contained also *débris* of peptic glands. Binz has

equally observed this elimination of iodine by stomachal mucous membrane in animals, to whom he had injected from forty-five to seventy-five milligrammes of iodine mixed with iodide; in all he recognized submucous ecchymoses.

Respired iodine.—The primitive proceeding of Barton and Piorry with the tincture on a plate, has been abandoned on account of the tracheal irritation and troublesome cough caused by it. The cigarettes and iodized tapers that have been employed in France are useless.

Pulverizations. Pneumatic apparatus.—When in contact with mucous membranes of larynx and bronchi, iodine produces great irritation and a troublesome painful cough. The advantage is this: if there are ulcerations, iodine, attaching itself to the mucus, forms a protection like nitrate of silver, under cover of which the ulceration may heal.

Respired iodide of ethyl.—The surest way of making iodine penetrate into the respiratory tracts and to determine the absorption of it is by the respiration of ethyl iodide, dose ten to fifteen drops several times a day. Huette recommended it in 1852, but this method was forgotten till in 1880 I applied it with success to the treatment of asthma. In phthisis it finds useful applications.

Applied iodine. Painting with tincture.—As a resolutive, tincture of iodine is frequently applied externally with such tenacity that the skin becomes damaged, cracked, painful, and the true derma is laid bare. People pretend thus to favour the resorption of subcutaneous effusions and even of tumours. How can this be a *resorbing* agent; by what method? Is it by derivation, revulsion, over-activity of vessels? These are but words; there are two ways of explaining it.

Mode of action of tincture of iodine.—1. Iodine is very prejudicial to living cells; if the tincture penetrates as far

as the neoplasm, this may, by the action of the protoplasmic poison, be stopped in the energy of its increase and be led to disappear; iodine will thus have favoured the resorption of the tumour. This explanation presupposes the absorption of iodine by the skin, its penetration to a distance, and its appearance in the urine. And is it so? Without doubt iodine is evaporated and absorbed by the respiratory tract, and the proof is that it passes into the urine (Demarquay). Further, it is, according to Röhrig, absorbed by the skin. He dipped his finger for fifteen minutes into the iodine contained in a bottle, hermetically adapted in the neck to the volume of the finger. Fleischer throws doubt on this experiment, and says that the iodine in such case does not appear in the urine.

2. The iodine determines a sort of erysipelas, of which the diminution corresponds with solution of surrounding tissues. Schede painted the skin of a rabbit with tincture of iodine; there resulted from it extravasation of lymphatic cells into the subcutaneous tissue, afterwards into the derma, into the intermuscular tissue, and even the periosteum. At the end of a week, after the diapedesis, a retrograde state occurred, fatty degeneration which attacked the neighbouring tissues. The epidermis is compromised, the derma is swollen, with white cells and dilated vessels; it is true factitious erysipelas. This explanation is far-fetched, and the fact itself of the disappearance of a neoplasm by painting with tincture of iodine is more than doubtful. If these applications have the least effect it is because they act as a slight sinapism, followed by the absorption *in respiration* of some particles of iodine. Formerly, people thought to make iodine penetrate the skin by means also of ointments, baths, iodized cotton, without forgetting the *sachats,* and the iodized vests.

All these methods in reality acted by evaporation of

iodine, and its absorption by respiration. In natural iodized waters the same law holds good.

Doses of iodine. Experimental doses.—We can inject with impunity into the blood of a dog, for each kilogramme of body weight, two to three centigrammes of free iodine, dissolved with twice the quantity of sodium iodide. This would be for a man of ordinary weight (seventy kilogrammes) a quantity from 1·4 grammes to 2·1 grammes. Beyond four centigrammes of iodine or two grammes of sodium iodide the dog dies in both cases in the same manner. At the end of four or six hours he exhibits general weakness, with respiratory trouble, which causes death after twelve to twenty-four hours. Sanguinolent pleuritic effusions, pulmonary œdema, and especially profound alteration in kidneys and urine, are to be noted.

Therapeutic doses of iodine.—The maximum dose of iodine has been little studied in man because of the habitual use of potassium iodide, which is in every way preferable.

Doses of potassium iodide.—I have seen it prescribed in a quantity, five to ten centigrammes daily, which made the object fallacious. The dose of fifty centigrammes daily has no action, that is why Trousseau failed in the treatment of asthma. We must for this disease increase rapidly up to two grammes: in phthisis to 1·50 grammes at least, unless there be hæmoptysis, in which case it is better to abstain from it altogether. In syphilis, according to the indications of Fournier, four, six, or even eight grammes. To avoid, or at least to diminish, the unfavourable action on mucous secretions, it is useful to add to it five to ten centigrammes of extract of opium daily.

Iodide of sodium.—In accordance with very incomplete notions of experimental physiology, iodide of potassium has been said to act in a dangerous manner on the heart in virtue of its potash. The fashion has been to substitute

sodium iodide, which would be harmless and without action on the circulation. As potash in doses of only a few grammes has never diminished the heart's action, and also the diffusion power of sodium salts is very much inferior to that of potash salts, the result is that we must double or treble the dose of sodium salt to obtain a real iodic saturation. These results have been obtained from experiments conducted by Bochefontaine in the laboratory of the Hôtel Dieu.

Absorption and elimination.—For the alkaline iodides absorption is the same as for metallic iodine, that is, it takes place in an immediate manner. After a few minutes iodine is to be found in the urine—a fact of which we may be assured by starch paste. The colour is produced when iodine is present in the proportion of one part to a million.

Means of elimination.—The elimination of iodine is effected by all the secretions: we shall see under what forms.

Urine.—The principal emunctory is the urinary liquid; it is in the urine that iodine is found in a few minutes, and where its elimination ceases at the end of twenty-four hours. Experiments have principally been made on the alkaline iodides, and definitely it is in the form of sodium or potassium iodide that metallic iodine quits the organism.

Saliva.—Elimination is slower by the saliva. Iodine may still be found at the end of three weeks, but it is to be noted that in passing into the saliva it is not rejected, it returns to the stomach, and consequently to the blood; in this way we may explain the reappearance of iodine in the urine.

Tears. Skin. Milk. Mucus.—Iodine is to be found in the tears, sweat, milk, and in a very definite manner in the mucus. The mucous membranes eliminate it easily, and the respiratory organs as well as the expectoration contain it habitually during medicinal use.

Intermittent elimination.—Whatever be the eliminatory liquid, we must note intermissions, times of stoppage, which mark all the period of purification; thus, during several days iodine fails in the urine, to be found there again later.

§ 137A. *Necrophytic Properties.*

Empiricism has long preceded experimental physiology, and has established and consecrated the superiority of iodine in the treatment of scrofula, syphilis, and tuberculosis.

Scrofula.—It was by analogy with goître that iodine was employed in the cure of scrofula; the results have exceeded anticipation.

Syphilis.—In its turn the apparent analogy of scrofulous lesions and syphilitic tumours was not long in leading to trials of iodine against syphilitic accidents; here, again, the triumph was complete. People thought themselves happy to be able to substitute iodine for mercury, which had been dominant for ages, and had, so to speak, monopolized all the therapeutics of syphilis.

Tuberculosis.—The third virulent malady which has been for half a century submitted to treatment with iodine is tuberculosis; we shall see what have been the results of this medical practice, and on what ground iodine acts in the malady. There are three chronic maladies which are within the domain of iodo-therapeutics. All three are of infectious nature. Scrofula and tuberculosis are characterized by the same virulent agent, the bacillus. Syphilis, of which the parasite is still to be found, constitutes the type of maladies transmissible by inoculation from man to man.

Chronic virulent maladies transmitted from the horse to man, namely, glanders and chronic farcy, have also been submitted to the action of iodine (Ladike, 1842; Payan, 1843; Cazin, 1854; Bourdon).

Acute virulent maladies. Anthrax.—It was in the treatment of anthrax that Davaine first showed the antiseptic power of iodine. "If we take," said he, "blood from a carbuncled fowl, and dilute it to 1000 or even 10,000, and then place in contact with it during fifty or sixty minutes a very weak solution of iodine, the guinea pigs that are inoculated with from two to four drops of this mixture continue to live, whilst other animals inoculated with similarly diluted blood, without iodine, infallibly succumb." We have seen men cured by the iodine treatment, the solution employed internally, and also injected into the œdematous skin as follows :—

Iodine, 25 centigrammes.
Potassium iodide, 50 centigrammes.
Water, 1000 grammes.

Typhoid fever.—Wildbrand has prescribed iodated iodine, and claims to have obtained considerable remissions of temperature. Liebermeister even believes in a diminished mortality from its use.

Diphtheria.—In this terrible virulent infection everything has at present failed. Local applications of tincture of iodine (Lecointe, Perron), preparations of iodine internally have remained without success, like all antiseptics, indeed, like all remedies in general.

Thus the action of iodine seems to be exercised only in certain microphytic maladies, particularly those which need for treatment a long impregnation of the organism with necrophytic and, we may add, trophic agents.

§ 137B. *Secretory Action.*

One of the properties of iodine which interests us most is its power of modifying the secretion and even the texture

of the mucous membranes and glands as it passes through them.

Slight iodism.—It may happen that the first impressions of the medicine on organs that eliminate it are painful, and there results a certain number of inconveniences which it is well to describe; I shall designate them as *petit iodisme,* as a distinction from the intoxication or iodic saturation. It is a collection of painful symptoms which often torments the patients up to the point of making them give up the treatment. Do not be afraid of insisting on the small importance of these incidents, which in no way indicate intolerance, and which are ordinarily dispersed in a few days, especially if we take care to add to the preparation of iodine a certain quantity of opium.

I. *Secretory effects. Vaso-conjunctival mucous membrane.*—The most constant and earliest phenomenon is coryza, with flow of tears, afterwards infection of the conjunctiva, with dilatation of the vessels. We find iodine in the nasal mucus, in the tears; all is explained by this iodic excretion.

Bucco-guttural glands and mucous membranes.—Many of the patients complain of constrictions at the throat, dysphagia, grating in the pharynx; the guttural mucous membrane is red, and often there is a slight infiltration of the uvula and pillars of fauces. It is *angina iodica.* The tongue is rapidly covered with a smooth grayish coat which may well be the result of the passage of iodized saliva over the lingual epidermis, and which in all cases causes a nasty taste in the mouth; this sensation is sometimes carried so far as to produce want of appetite.

Laryngo-tracheal mucous membrane.—This is much more tardily and more rarely affected than the superficial membranes. It is only by prolonged use of the medicine or by exaggerated doses that we observe hoarseness of the voice, cough, or anxiety. In general the pharmaco-dynamic

effects are of good omen; these are more marked secretion, greater consistency of mucus, an easy cough, and especially free respiration which astonishes all the patients. It is only in grave iodism that we note hæmoptysis and œdema of the glottis.

Digestive organs.—Whether the iodine has been introduced directly into the stomach, or that it has arrived there after being absorbed by respiratory passages, or by the rectum, the digestive functions are little modified; if in certain exceptional circumstances the *appetite* has been increased, it is when one administers insignificant doses, as a few drops of tincture or a few centigrammes of alkaline iodide. If iodine has been given to stop vomiting, it is still due to this minimum dose, for in general a full dose produces no effect, and in toxic doses it causes sickness.

Skin and cellular tissue. Acne.—Habitually iodine *provokes* in the first few days the appearance of simple acne on the skin of the face or body, and sometimes erythematous stains which are not effaced,—all the other cutaneous lesions, such as œdema of the visage, purpuric spots, sanguinolent pustules or bullæ, urticaria, enter into the order of grave iodism and deserve to be taken into consideration; it is a warning; we must stop.

Kidney and urine.—Iodine does not act on the quantity of urine, but on its composition and on the renal tissue. *The kidney* is formed of acid tissue which sets iodine free under whatever form it be found, and the metal determines an *albuminuria,* or rather an albuminous nephritis, which has been noted even after simple painting with tincture of iodine, particularly in children (J. Simon).

II. *State of iodine in the secretions and eliminatory organs. Free iodine.*—After the introduction of iodides into the economy, free iodine appears in the organism, but only in certain parts; thus, according to Bucheim aud Sartinon,

it is free iodine which determines catarrh of gutturo-nasal mucous membranes, and even the cutaneous eruptions which are observed so often during the administration of potassium iodide.

Guttural mucous membrane.—The iodine set at liberty comes from the iodides which appear abundantly in the saliva, and which find themselves in contact with quantities of CO_2; this gas acts on the iodides and nitrites, which are never wanting in these regions. We know that solution of starch and potassium iodide, which contains a nitrite, always turns *blue*, when one passes a current of carbonic acid through it; this colouration is due to free iodine.

Skin.—Here the iodides are doubtless decomposed by the acid contents of the sebaceous and sudatory glands; hydriodic acid is formed, which easily liberates free iodine, causing the appearance of eruptions.

Stomach.—It is doubtless also the acid of gastric juice, which acts in disengaging iodine from potassium or sodium iodide. The metalloid has not been found in a state of purity, because it combines rapidly with albumen, and thus escapes direct demonstration.

In the parenchyma.—We suppose that iodine becomes free in the blood and parenchyma of tissues, and acts especially on the vascular walls, irritating them as in direct application; in reality the method of elimination of iodine after the use of potassuim iodide seems to indicate the process. Whilst the greater part of the iodine is rapidly eliminated by urine, saliva, sweat, and other secretions (Lewald), one finds still, during entire weeks, traces of it in the saliva when it has vanished from the urine (Cl. Bernard). This fact is explained if one admits the presence of iodiferous albuminates in the organism, which are only capable of passing into secretions, which, like saliva, contain albuminoid elements.

Iodo-albuminates in parenchyma.—But it is not demonstrated that the alkaline iodide has a particular affinity for the albuminates of the organism, and acts by their intermediation. Binz believes that it is free iodine which acts everywhere, and that it is the histological element, *protoplasm*, which sets it at liberty. Protoplasm stimulates the properties of oxygen which exists or abounds in these tissues; but the cells of animal tissues do not liberate iodine from its compounds except in presence of carbonic acid, and as our bodies furnish marked quantities of this gas, it is then the acid that decomposes potassium iodide, "so that there results potassium bicarbonate and hydriodic acid, the latter being immediately decomposed by this ozonised oxygen, it being understood that the affinity of hydrogen for oxygen is much more considerable than for iodine" (Binz).

Iodine in the urine.—Iodine appears in the urine, with alkali as an iodide, which is easily explained, for iodine being set free in certain tissues, is taken up again by the alkalies of the blood; it continues thus to progress and metamorphose itself, until at last, in the kidneys, it is found being eliminated as a salt, whilst another portion of iodine remains free, and passes so into the urine, on account of the acidity of renal tissue. It is because of this incessant transformation of iodine into iodide that potassium iodide is harmless; it must not be considered as a poison (Binz).

III. *Application to tuberculosis.*—The secretory effect of iodine, on respiratory mucous membranes is very remarkable in individuals attacked with dry bronchitis, asthma, and especially with dry catarrh, which marks the commencement of tuberculosis. When consulted on the subject of a dry paroxysmal cough, with or without vomiting, and especially without expectoration, in most cases you would not be able to find a *râle*, either dry, sonorous, or sibilant.

If iodine be administered, you will be struck with the appearance of three phenomena after a few days: first, certain *râles* are to be found at the apex of the lung, which is but a mediocre advantage; secondly, a much greater facility of respiration; and, in the third place, a more easy cough, the expectoration being more fluid. What has happened? Under the influence of iodine the bronchial secretion augments and ceases to be concreted, so that the air enters forcibly, and penetrates more profoundly; the cough is now exerted on a true mucous secretion instead of being unavailing with spasmodic character. These are real benefits for the patient.

Secretory effects on bronchial mucous membrane.—Iodine produces on the secretory function a true excretion of morbid products, which are found accumulated in the mucous cavities. As it is allowable to suppose that the parasites occupy the mucous membrane or the subjacent tissue, they may submit to elimination with the products of expectoration exaggerated by iodine.

General eliminatory effects.—These ideas have been pushed too far; people have attributed to iodine an eliminatory power much more marked and more general than it really possesses. It is on this function that the treatment of certain metallic intoxications has been based, as mercurialism, saturnism. The idea was expressed in the year 1844 by Melsens and Natalis Guillot. Lead alters all organs, all tissues, so profoundly that it has been necessary to renounce this means for some way of detaching metallic particles. Mercurialism is still often treated with iodine, but with very doubtful success.

Conclusion.—Does iodine act in purifying the blood from the bacilli which it contains? Nothing leads us to think so; and everything tends to show that, as blood scarcely ever contains developed bacilli, the eliminatory

power is less exercised on the blood than on the respiratory mucous membranes, which are the habitual seat of bacilli in pulmonary phthisis.

§ 138A. *Iodine in the Blood. Its Effects on the Blood. Oxydizing Action.*

State of iodine in the blood.—The action of iodine on the blood and tissues, on account of its known affinity for hydrogen, is shown by the formation of hydriodic acid, at the same time that a destruction of molecular framework of tissues is effected.

Albuminoid compounds.—Another affinity not less marked is that of iodine for the albuminates. We know that starch-paste, coloured blue by iodine, is decolourized by albuminous solutions; for example, the serum; what is formed is an albuminous iodate. As this iodo-albuminous compound is not at all stable, it is destroyed easily by the coagulation of albumen or by dialysis. The alkali of albuminous serum is not saturated by the iodine that is added to it; but neutral serum, or serum deprived of salts, becomes *acid* by the addition of iodine.

Iodide and iodate.—When the iodo-albuminate is destroyed by coagulation or dialysis, the alkali set free from the albumen is combined with iodine in two fashions; to wit, as much iodide as iodate. What proves it is this: if one adds pure hydrochloric acid to a solution of iodide, the solution which is very acid remains colourless; but if to the iodized serum one adds the same acid, it turns yellow from liberation of iodine. Also, as iodine always meets an alkaline carbonate in the serum, it is transformed into iodide and iodate. These two salts circulating side by side should in an *acid* medium give up free iodine, which, in being developed, acts on the tissues in an irritant manner. We really know that a mixture of iodide and iodate, but

only the mixture, is instantly decomposed by hydrochloric acid; as a result a certain quantity of iodine is set free. The same experiment succeeds with the gastric juice (Rabuteau); so that iodine probably becomes free in the stomach.

Action on hæmoglobin.—Hæmoglobin as well as serum sets a large quantity of iodine free without losing its properties. The corpuscles neither increase nor diminish in number, although hæmatigenic properties like those of iron have been attributed to it.

Action on fibrin.—It has been supposed that iodine acts on fibrin to fluidify it, but this is a preconceived notion.

Oxydizing properties of iodine and potassium iodide.—As iodine and potassium iodide are endowed with marked denutritive properties, which is shown by the atrophy of a large number of morbid products, tissues, and healthy glands, people have supposed that the materials of denutrition pass into the urine, under the form of urea, of which the quantity, therefore, will be more than normal. But, according to the experiments of Rabuteau on himself, it is the contrary which appears to be true. After a week of uniform diet, he took for five days, one gramme of potassium iodide, and observed that the normal proportion of urea, which had been from 24·6 grammes to 21 grammes, fell gradually down to 19·30 grammes, and even as low as 13 grammes. This last proportion was obtained five days after leaving off the medicine. Thus iodine moderates the progress of denutrition. Fubini has stated the same facts, whilst Beck, who has examined syphilitic patients, has not found any modification in the proportion of urea after 1·50 grammes of iodide had been administered during six days. But further; is the proportion the same, or are there variations in dif-

ferent pathological conditions? It is proved that in certain morbid states urea is eliminated in greater quantity under the influence of iodides. Bouchard has observed this phenomena in diabetes; but as in this malady urea is nearly always in excess, we may suppose that there has been increased excretion, without increased formation of urea; it is then as an eliminator that it produces augmentation of urea in the urine. In other morbid states, general nutrition is improved in an evident manner; is this only by curing the malady and hindering fresh neoformations? We see it manifestly in syphilis and in scrofula; the patients are cured and fattened so that one asks if this hypernutrition bears on the albuminous tissues or only on the fat; though it is in no way demonstrated to be shown by excess of urea. If this phenomenon occurs, one has also the right to ask if iodine in small doses does not influence the stomach favourably, augmenting the appetite, digestion, and consequently assimilation of nitrogenous principles, whence there results an excess of urea in the urine, for we know that the urea increases in direct proportion to the quantity of nitrogen in the food.

§ 138B. *Atrophic Actions.*

Local atrophy.—People have discussed the oxydizing properties and the denutritive power of iodine, but they must not be confounded. The denutritive action of the remedy, which is not doubtful, is shown by the atrophy of certain morbid tissues; it acts on some glands in the same way with normal conditions, to wit, the lymphatic glands, the thyroid gland, the female mammary glands, and the genital organs of man; but this is not demonstrated for moderate doses. Further, when it is long employed, and in larger doses than two grammes, it ruins the organism by producing grave iodism.

Gravé iodism.—One sees the pulse accelerate if previously calm (Küss), rest at the same point if it were accelerated, and become full and ample, afterwards smaller and depressed. The skin becomes the seat of acne, erythema, and of papules, which are not effaced by pressure with the finger; that is, sanguinary suffusions, ecchymotic marks, and even *erythema nodosum* (Talamon has observed a case of this kind). The eye is congested, and becomes the seat of grave conjunctival inflammation. Angina iodica shows itself in the form of an exudative inflammation with œdema of the uvula. The broncho-pulmonary mucous membrane is affected in its turn with a catarrhal secretion; there is dyspnœa and often hæmoptysis of slight intensity, which must be distinguished from hæmoptysis of tubercular origin.

The ovaro-uterine mucous membranes are congested; it is not rare to observe a return of the periods in chlorotics; in them hyperæmia replaces local anæmia of genital organs. It is to be remarked that iodism is easily produced in certain therapeutic or pathological conditions. The simultaneous use of alkalies and iodine constitutes, in my experience, a very favourable condition for iodism. I have seen cardiac patients and asthmatics, who for several months took two and a half grammes of potassium iodide daily without inconvenience. A course of Carlsbad or Vichy, which patients prescribe for themselves for certain gastric troubles, will often determine grave iodism, whilst in general asthmatics and cardiacs support iodic medication very well.

Applications to the therapeutics of neoplasms and maladies by lessening nutrition.—Without going as far as iodism, we may obtain atrophic effects on certain neoplasms or certain normal states resulting from diminished nutrition (Bouchard).

Neoplasms.—Abstraction being made as to syphilitic and scrofulo-tubercular neoplasms, iodine is without action on tumours called malignant, as cancer; of doubtful action on benign tumours, as fibrous bodies; and very variable as to goître.

Goîtres.—There are goîtres which are not in any way modified by iodic treatment. Acute epidemic goîtres resist; exophthalmic goître still more so; and as to goître endemic in the Alps, it appears to constitute a particularly favourable condition as to iodism.

Polysarcia and diabetes.—Here iodine acts most favourably. My practice is explicit in this matter, but there is a limit and an inconvenience. If you associate iodine with an absolute Banting *régime,* depriving your patient of fat and feculents together, strength is lost as well as corpulency; the loss of flesh will be achieved though with considerable waste of force. If, on the contrary, you prescribe training *régime,* that is, alimentation by albuminoid substances and fresh vegetables, without prohibiting fat, you may, taking care not to exceed small doses of potassium iodide, obtain a favourable result. Avoid excessive doses and exclusive dietary. It is the same in diabetes, which requires much more care. As there is between glycæmia and tuberculosis an intimate connection, we must learn how to prevent and how to treat diabetic phthisis.

Special atrophic action of potassium iodide.—Its principal use is directed against neoplasms in general, especially the syphilitic, inflammatory, glandular, etc. Its action in this case, without being infallible, cannot be contested; the very varied facts permit us to conclude that cure does not depend on a special or specific action of the salt on organs or on given systems, but that it is the result of changes in nutrition and oxidation in general. These modifications

have not the same necessity to be shown in a marked manner with normal conditions; for example, by the increase or diminution of urea during a shorter or longer time. More or less modified oxidations signify simply this—that the less stable pathologic products alone are brought within the domain of denutrition. The iodide determines also a series of actions which do not belong to any other salt. It is very rapidly absorbed; it penetrates with great facility into all tissues, and in presence of sodium chloride it is decomposed into sodium iodide and potassium chloride; consequently its saline action should be considerable. If we consider, further, that by the side of the action of potash, and that not less marked of its iodide, both being shown in the denutrition of certain organs, that we may add a direct effect on the organs by iodine set free, we can understand the efficiency of potassium iodide. It is probable that all the chemical elements contribute to it, for in the treatment of indicated morbid conditions it cannot be replaced by any other iodide, nor by any other potash salt, nor in general by any easily assimilable salt.

Dose.—It is certain that animals support a dose of three centigrammes per kilogramme of body weight, and that man may take two or three grammes daily without having the least digestive trouble or the least uneasiness.

Comparison with sodium iodide.—Whilst potassium iodide, in presence of sodium chloride, is transformed into sodium iodide and potassium chloride, what becomes of ingested sodium iodide? Does a double decomposition take place, in presence of the potash salts, of blood corpuscles? If it is thus, we reconstitute potassium iodide, and one asks, What is the superiority of sodium iodide over the potassium salt? We know also that the dose of sodium iodide must be singularly high to enable the iodine to act.

§ 139. *Iodine against Dyspnœa.*

We have come to the most curious property of iodine, its respiratory function, which I made known in my academic memoir of July, 1879.

History of anti-dyspnœac properties of iodine.—My first researches were on asthma. Whatever its nature, simple, gouty, herpetic, intermittent, or permanent, I found under the influence of iodine an immediate disappearance of attack, and the return of the emphysematous lung to the normal condition. Secret remedies containing iodine had been extolled; very small doses of iodide had been indicated by Trousseau and Jaccoud, but afterwards abandoned on account of insufficient action. My experience was with iodic medication resolutely conducted, the daily dose being two or three grammes, often without any addition, though with rigorous continuation of treatment, the anti-dyspnœac effects were constant, and without fail. Gradually and logically I was led to attempt its use in cardiac dyspnœa, and to give it in pulmonary phthisis.

Since 1879 I have been able to collect nearly two hundred observations relative to simple or gouty asthma, the intermittent or permanent form, and without meeting a single case which has resisted iodic medication. To judge from the prescriptions of my earliest opponents, I see with satisfaction that their scruples have vanished, and that their theoretical wish to preserve a diathesis for asthma, which I declared curable by iodine, has completely ceased before the evidence of facts. Opposition is no longer as to the kind of treatment, but, what is more grave, as to the methods and doses of the medication; this is the origin of numerous and vexatious failures.

Respiratory action of iodine.—In all asthmatic forms of dyspnœa, iodine exercises a double action—that is, as

a modifier of bronchial secretions, and as a respiratory medicament. The mucosities become more abundant, but also less consistent, so that the obstructed respiratory tubes become more permeable to inspired air. That is not all; iodine produces increased activity in the respiratory nervous centre and in the bulb, which singularly facilitates the reflex act of respiration, that is the impression received, or the sensation of need of air, and its transmission through the spinal cord by nervous influx on the motor nerves of respiration, particularly the phrenics. This is proved by all the patients noticing freer respiration, which subjective effect never fails. On the other hand, the synergy of the respiratory muscles is manifest and prompt; the fuller ampliation of the chest, especially in a longitudinal sense, that is by contraction of diaphragm, cannot be in the least doubted. It remains to be seen if this activity of the medulla is primitive, or if it is the result of a more marked afflux of blood than in normal conditions.

Applications to cardiac dyspnœa.—The constant respiratory effect in bronchial dyspnœa led me naturally to the application of iodine to cardiac dyspnœa, and to maladies of the myocardium itself. This is not the place, however, to insist on this subject.

Applications to tuberculosis.—Here we find, so to speak, all the useful properties of iodine united. Without speaking of the microphytic effect or its saving action, I point out the secretory power that iodine exerts on the bronchial mucous membrane, and especially the modification of respiration in tuberculous patients at all periods of the malady. The only inconvenience is the possible production of hæmoptysis; the only contra-indication is perhaps fever. The explanation of blood effusions is easy to be found.

§ 140. *Neurotic, Nervo-vascular, and Hyperæmic Actions of Iodine.*

Iodine gives increased activity to general circulation.—This effect has been noted in experimental iodic intoxications; acceleration of pulse has also been noted in accidental poisoning (Küss). Local hyperæmia is still better demonstrated; primarily, congestive action on glands has been remarked; this leads to hyper-secretion, then the ovary becomes the seat of hæmorrhage, and there is congestion of mucous membranes in general when they act as secreting organs. Claude Bernard has shown that no function is made more active, no secretion is produced, without physiological hyperæmia. This is the cause of the suffusions of blood that are found in the bronchi under the influence of iodine; it is its principal disadvantage.

Iodic hyperæmia is of the nervo-vascular order. Physiological hyperæmia, and still more so that provoked by poison, is under the influence of the vaso-motor system, which presides over secretion, that is through the blood.

Cerebro-bulbous hyperæmia.—If, then, the vascular nerves are brought into play, so as to render the vegetable functions more active, have we not good right to suppose that the circulation in brain and medulla is over-excited by the same mechanism under the influence of iodine? What is called iodic intoxication appears to be the proof of it. Binz has also described a sort of narcotic iodism, which recalls this drunkenness. If, then, the brain is the seat of increased functional activity on account of more rapid blood-circulation, there is no reason that the medulla should not submit to the same over-activity; and this is shown by and through respiration.

Direct neurotic action.—If it is not thus, we must admit, with Binz, there is localization of iodine in the central

nervous system; and, in fact, Vulpian has demonstrated that iodoform invades the brain and its dependencies in an evident manner. The mechanism of the action of iodine on the nervous system is, then, a subject of discussion; but the fact remains as acquired for science.

§ 141. *Iodoform.*

I. *Chemical characters and preparations.*—Iodoform, CHI_3 is, of all iodic preparations, the one which is nearest to free iodine, of which it contains 96·7 per cent.; it is still more nearly allied by its composition to chloroform, whence the name of iodoform given by the immortal chemist, Dumas. Nearly insoluble in water, soluble in fifty parts of cold alcohol, in ten parts of warm alcohol and five parts of ether, iodoform is most readily dissolved by fat. When dissolved in fat or alcohol it does not yield iodine, except in presence of light and oxygen. In wounds, for the treatment of which it is often employed, the conditions are fulfilled, oxygen being found constantly in the oxyhæmaglobin; the first condition, light, is replaced in living tissues by the cells themselves, which are sufficient to disengage iodine.

II. *History and doses.*—The first pharmacological essays on iodoform are due to Cogswell, 1837; Righini, 1863; Kendrik, 1874; Binz, Högyes, Behring. Mosetig and Mikulitz, in 1880, substituted iodoform for carbolic dressing, but numerous failures and mortal intoxications made them abandon this method. The poisoning was shown by anguish, amnesia, hallucinations, vomiting, hæmaturia, death by pulmonary œdema. On necropsy, fatty degenerations of heart, liver, and uriniferous tubules were found. Rummo, who made these researches in Vulpian's laboratory, has further described grave lesions of brain and spinal cord. In medicine it is employed in moderate doses, as

twenty to fifty centigrammes daily, in the form of pills, and with the addition of a deodorant, as coumarine; in larger doses than 1·5 grammes daily, iodoform may become toxic. It is in the treatment of three specific maladies, syphilis, scrofula, tuberculosis, that iodoform presents advantages. The Americans first of all used it for neuralgia and in malaria. Moleschott was the first who (1878) used it in *phthisis*. He treated renal leucæmia, effusions into serous cavities, glandular enlargements, in the same way.

III. *Mode of action on histological elements in general.*—Whilst undissolved iodoform acts as an inert body in presence of bacteria of putrefaction, we know that free iodine is a most energetic anti-parasitic. The iodine, which is gradually disengaged from the iodoform, is opposed to the septicity of the wound, and hinders emigration of leucocytes by paralyzing protoplasm, and, by avoiding the products of decomposition, favours the production of healthy granulations.

Action of iodoform on tubercular cells.—According to the curious experiments of Marchand (Arch. f. Path. Anat. Berlin, xciii., September, 1883), iodoform seems to act in a special manner in hindering the formation of certain histological elements of tubercle. In artificial tuberculous wounds of the cornea we recognize the parts treated with iodoform, in that they are formed solely of pus corpuscles, which present a very pure red colouration when treated with picrocarmine, whilst the fragments which were not treated with iodoform, contain further a great number of epithelioid cells, which present a yellow colouration. With iodoformic treatment the formation of giant cells is entirely avoided.

Combinations of iodine with albumen.—But is the action of free iodine direct, or does it pass first into the form of

albuminate, as Högyes believes? It is certain that iodic albuminate is decomposed by simple dialysis, and is transformed into iodide and iodate, which are decomposed in their turn.

Affinities of iodine with different elements.—Free iodine may be combined with the hæmaglobin of blood, or with the uric acid of urine.

IV. *Physiological effects on circulation and innervation.*—In doses of from three to ten decigrammes, iodoform determines, according to the experience of Rummo, a slackening of the pulse; at two grammes a diminution of blood-pressure; but these phenomena do not last. The nervous centres, and later, the peripheric nerves, lose their excitability, which is soon, however, replaced by convulsions.

State of iodine in the urine.—In all cases the iodine formed is not eliminated as such, but is united to alkalies to pass principally as potassium iodide (Harnak and Grundler). These physiologists only once found free iodine. Rummo has never found it, but he thinks, with Vulpian, that iodoform is eliminated as iodate. They have remarked that the elimination lasts several days, even where there has been but a single ingestion of iodine. Zeller has made the same observations, and has further described a true accumulation of iodine in the blood of the animals experimented on, so that the resorption by the blood and the elimination by urine do not keep parallel.

V. *Therapeutic results in the treatment of phthisis.*—The observations of Moleschott attracted the attention of the Italian school, represented by Semmola, afterwards by Ciaramelli, De Renzi and Fasano; and lastly, of the Vienna school, where the works of Professors Schnitzler and Drasche threw light on this iodoform question, but without settling it in a complete manner. According to the Italian authors, iodoform acts especially in the first stages of phthisis, which

may thus be cured. In advanced cases the remedy may equally be very useful in lessening expectoration, paroxysms of cough, fever, and in preventing the process of caseation; in other words, by prolonging the life of the patients. In Italy iodoform is given in pills from four to seven centigrammes, with extract of gentian, or it may be used in the form of inhalation or liniment (L. Spallanzani). At Vienna the interesting discussions raised at the Society of Physicians may be thus epitomized:—

1. Simple ulcerous or tuberculous laryngitis, which precedes or accompanies pulmonary phthisis, is marvellously modified by this means, especially when employed in the form of fumigation (Schnitzler); laryngeal pain and dysphagia rapidly disappear.

2. Diabetes (according to Moleschott and Semmola) may be cured entirely by this method, which is superior to salicylic acid, and rapidly reduces thirst, secretion of urine, as well as the production of sugar; but Drasche, of Vienna, considers the amelioration as but temporary.

3. Phthisis has been treated by Schnitzler with inhalations of an ethereal or alcoholic solution of iodoform, and also cod liver oil, containing iodoform, prepared in capsules; the results were negative. Seventeen cases of phthisis were treated by Drasche and Muller with pills containing two centigrammes of iodoform and extract of gentian. Out of seventeen patients, eight increased in body weight, after from three to six days; in a second series the results were less favourable. Its action on the temperature was most often injurious. Kowalski considers the remedy as useful, but only when it is in contact with tubercular surfaces; thus, in tubercular emphysema he had not obtained the least advantage. In a general manner what strikes me in these observations, and in those which I have been able to collect, is the very marked utility of iodoform

in curing pectoral pains, neuralgias, myosalgias, which so often afflict the phthisical. From two to four decigrammes in pills will suffice to attain this end.

XLII.

ARSENIC.

In certain respects arsenic has analogous properties to those of iodine, but it differs in many ways. We shall find here:—

1. If not anti-parasitic at least anti-fermentative properties.

2. The action of the medicine as a proven means of diminishing expenditure, and especially as a powerful modifier of parenchyma.

3. The respiratory or anti-dyspnœac function, which is most evident.

4. Arsenic produces a most marked diminution of the pressure of the blood in the vessels, and this profound modification of the circulation will serve us as an interpretation of certain phenomena which have remained obscure despite their importance.

5. It is perhaps thus that we can explain the favourable effects of arsenic on the nervous system. Arsenic differs from iodine in an absolute manner, because it does not produce any appreciable change in the secretions of mucous membranes at the moment of its elimination. It differs from iodine especially in producing a serious injury to the digestive organs, either by its direct action on the stomachal mucous membrane, or following its elimination by the stomach after having been absorbed.

§ 142A. *Absorption. Elimination.*

Preparations employed.—The most useful preparations are arsenious acid, arsenite of potash, arsenite of soda, arsenic acid, arseniate of soda.

(a) *Arsenious acid.*—The therapeutic dose of this acid should not exceed five milligrammes, gradually and by exception it may be increased up to one centigramme. The large doses which have been used for experimental toxicology find no application in clinical medicine except to explain the mechanism of medicinal actions. Arsenious acid has been employed in granules of one milligramme; they have the grave inconvenience of accumulating in the intestine, or, if they are hardened, of passing into the evacuations. It is preferable to use arsenious acid in solution, although it only dissolves in a large proportion of *hot* water. The formula of one gramme to 1000 of alcoholized water is the most used.

(b) *Arsenite of potash* (Fowler's solution).—The French liqueur de Fowler contains one part of arsenious acid in 100, and is consequently stronger than the English preparation, which has one part of arsenious acid to 120. The dose can rarely be allowed to exceed fifteen drops with impunity; a diluted aqueous solution of the arsenite has been used for subcutaneous injection (Köbner).

(c) *Arseniate of soda.*—The dose of crystallized salt is from one to two centigrammes in solution.

(d) *Arsenic and sulphides of arsenic.*—The doses indicated above are the *extreme* limit for therapeutic use, although people talk of being accustomed to larger doses. In Styria and Lower Austria there exist arsenic eaters, who, to sustain their strength, facilitate respiration, and to develop muscular action in the ascent of mountains, take

two or three centigrammes at a time; the women and young girls also take this poisonous dose to heighten their colour, and get an appearance of robust health. We are uncertain as to the preparation employed—whether it is metallic arsenic, which is much less dangerous than arsenious acid, or it may be sulphide of arsenic, which Huseman considers nearly inoffensive. We can learn nothing with scientific accuracy from these legendary statements. It is unknown how much of the poison is absorbed, and how much passes unabsorbed into the fæcal matters.

Degrees of poisonous action of different arsenical compounds, and their effect on the stomach.—We must distinguish the ulcerative action of arsenious acid which remains in an insoluble state in the stomach from the gastric affection which results from the action of blood impregnated with the poison. The arsenical compounds absorbed by the blood most probably act in an identical manner, and to determine the poisonous influence of each compound, we must especially know to what degree it can be absorbed. Arsenic acid is less poisonous than As_2O_3; it only acts on the lower part of the digestive tube after being converted into arsenious acid.

Metallic Arsenic is not dangerous unless it contains As_2O_3, or is converted into arsenious acid in the intestine (Schroff). The sulphides (orpiment, realgar) remain intact in the intestine and are not poisonous—at least, unless mixed with arsenious acid. In fact, it is this arsenious acid which seems to be the true poison, although it is far from acting as dangerously on the herbivora (horses, cows, sheep) as on man.

Absorption and diffusion.—After a very tardy feeble action on the digestive organs, arsenious acid enters the blood, then acts by its intermediation on distant organs, and profoundly modifies their oxidations. This diffusion is such that, according to modern experiments, we can and ought

to consider certain actions called local, as secondary results from arsenical blood; this often happens for the stomach. The absorption of arsenic is, in fact, so rapid, that even in acute poisoning we observe only but general phenomena. It follows that arsenic should be considered an enemy to all living tissues, on which it acts in the most deleterious manner whatever be the way of absorption.

Gastro-intestinal absorption. — It is by the stomach that the poison is most habitually introduced and absorbed, either as a medicine or as a toxic agent.

Arsenical mineral waters.—We must refer the action of certain mineral waters, whose composition has been long doubtful or unknown, to arsenic. In France there are a good number of springs which contain traces of arsenic. Only those containing an appreciable quantity can be taken into account, as *La Bourboule*, at the same time containing iodides and chlorides; *Mont Doré*, which is very feebly mineralized; *Royat*, which possesses a ferro-arsenical spring, and another one alkaline, after the fashion of Ems.

Subcutaneous injections.—Köbner has employed arsenite of soda in subcutaneous injection, but without any obvious advantage. The inhalations and arsenical cigarettes used by Trousseau have fallen into a just oblivion.

§ 142B. *Preservative Properties of Arsenic.*

Antiseptic properties.—It has been remarked that arsenious acid preserves corpses, and destroys up to a certain point the fermentation due to organized ferments. Sawitch and Johansohn have proved that fermentation may be stopped in the first two days, but on the following days it takes its course; so that if, for example, we deal with saccharine fermentation, the difference between arsenical solutions of sugar and those that are pure disappears on the fourth day. On the other hand, yeast left for *a long time* in contact

with As_2O_3 loses its ferment-producing power, especially starting from the moment when putrefaction commences under the influence of bacteria which are developed in it. Arsenic is endowed with anti-fermentative and disinfectant properties.

Is it in this way that arsenic acts on the parasite of marsh fevers? or on the supposed ferment of diabetes? Both are questions to be answered.

Preservative action against bacilli. — In a series of memoirs, Buchner has started a new theory of obtaining immunity from infectious maladies, particularly tuberculo-bacillary infection. His agent is arsenic, which is destined not to destroy the parasite, but to render the organism invulnerable by provoking a salutary inflammatory reaction, so as to constitute a bad culture-ground for bacilli. The important point is consequently to judiciously recognize the affected organ. Arsenic, phosphorus, antimony, are alike in producing with minimum doses a general inflammation of tissues which paralyses its deleterious action *wherever* the parasite acts.

Arsenic, Buchner says, has alone given proof of benefit against malaria, against adenoid scrofula. The arsenic consumers, who soon become slaves to the poison, like the Styrian arsenic eaters, preserve their organs from the action of the parasite. Thus is established a struggle between the cells of the tissues on the one hand, and the bacilli on the other. Then, as the parasites present an unsurmountable resistance to chemical agents, we cannot hope to find a parasiticide powerful enough to annihilate their effect without compromising the infected organ. If, on the contrary, we influence the organ, suppose the lung, so that it will resist the attacks of the poison, we shall have both a prophylactic and curative action on tubercle with the condition always of continuing the treatment long enough

to shelter the patient from all hurtful attacks of the parasite.

Observations of Buchner.—Full of enthusiasm for this theory, Buchner administered 2 c. c. of solution of arsenious acid 1 to 2000 (1 milligramme). Six patients were treated, and he came to the conclusion that arsenic acts surely against hectic fever, against the diffusion of the poison in the economy, finally against the destructive process of the lung already attacked by the parasite. The panacea for phthisis seemed to be found.

Observations of Kempner.—In twelve cases observed in one of the Berlin hospitals, Kempner seems to support the facts described by Buchner, but with grave restrictions.

(a) *The general condition*, said he, is ameliorated with regard to comfort; but there is no diminution of fever, and arsenic cannot be considered as an antipyretic like kairin. (*b*) The *sweats* generally diminish, but much less completely and quickly than when atropine is used. (*c*) The *appetite* is constantly increased and maintained in such fashion that alimentation, being possible and even easy, the patients increase in weight. This has happened ten times out of twelve. In a word, it is the best *corroborant* for phthisis, the most certain of the hygienic agents, which are only available in small numbers.

Observations and objections of Stintzing.—The researches of Stintzing at Ziemssen's *clinique* singularly changed the tone of these first predictions. With sixteen patients, who took from two to ten milligrammes of arsenious acid in solution, and of whom two succumbed, it was noted—

(*a*) That the temperature was lowered temporarily in three cases, remained the same in nine, augmented in one case, and left three other patients in the anterior apyretic condition. (*b*) The pulse and respiration, which were ordinarily increased, were only lowered twice out of sixteen.

cases. (*c*) The body-weight noted in thirteen patients became less in six out of thirteen. (*d*) The vital capacity observed in twelve of the cases increased four times, diminished seven times, and once remained stationary. (*e*) Out of fourteen cases with marked physical signs, they were increased in ten, and remained stationary four times. In no case was the morbid process arrested. Thus the effects of arsenic in phthisis may be considered as *nil*, and certain favourable cases attributed to the natural course of the malady. I shall make an important restriction as to the sub-oxydizing action, that is, the *saving* action of this medicine, which constitutes one of our most precious indirect tonics; one of the best trophics, particularly in chronic phthisis.

Observations of Thilenius.—This physician, who has care of consumptives at Soden, has for twenty years made use of arsenite of soda, dose two or three milligrammes daily, and has never seen anything but amelioration of general condition, often with surprising diminution of dyspnœa, and disappearance of nervous troubles, but without influence on the phthisical process, or on the bacilli.

Diverse observations.—Arsenic, says Leyden, does not possess any anti-bacillary property, and enjoys, at most, favourable action on nutrition. Buchner himself does not advise arsenic as an antiseptic, but as a means of fortifying living tissues, so as to deprive the bacilli of a nutritive soil.

§ 143. *Sub-oxidizing Action.*

Before studying this saving action of arsenical preparations, we must know in what state and condition arsenic is found in the blood.

State of arsenical acids in the blood. Relations with tissues.—By analogy with a certain number of metallic poisons, people have supposed that arsenic, once in the

blood, contracts chemical combinations with organic substances, either of blood or of ambient tissues, particularly with albuminoid bodies. It is thus transformed into albuminate, which will determine the destructive action of arsenic. Kendall, Edwards, Herapath, have proved by their researches that no one has been able to modify albuminates by arsenious acid. It is without any influence on the decomposition of these substances, and even without action in the stomach itself, where, in spite of direct contact, it changes neither the albumen, peptones, nor acid reaction of the gastric mucous membrane (Schäfer and Bohn). In presence of this unsolved difficulty we ought to inquire if arsenic acts on the gaseous elements of the blood, or in its turn is influenced by these gases. In a series of memoirs, Binz and Schultz have sought to demonstrate this singular theory, that arsenic does not act as such on the body elements, and that the atom arsenic, contrary to the atom lead or phosphorus, becomes toxic only by constantly *harassing* the blood and tissues to take up oxygen or to restore it to the blood. Arsenious acid, in contact with living cells, is oxydized and transformed into arsenic acid; this latter in turn is reduced to arsenious. When this chemical process is constantly repeated in the cells, oxygen constantly in the nascent condition finishes by gradually burning and destroying the bodily elements. Tissues which possess the most considerable nutritive and denutritive power are most violently attacked. In the blood, says Schultz, there is but a single movement of oxygen, that of reduction; whilst living protoplasm provokes with arsenious acid a double movement in inverse sense.

Dogiel considers the mutations of these two acids as both illusory. Harnack thinks with reason that arsenic, combined with other metals, acts as arsenic on the body tissues, and that all active arsenical compounds take the

same form in the body. At first sight it appears that arsenious acid will unite most easily with albumen. Formerly arsenic acid was considered most poisonous. But we do not know yet what are the affinities of arsenious acid for a given bodily element. We know only that arsenious acid leaves the blood and other liquids in appearance unaltered. It does not produce the least immediate functional trouble in the regions attacked. Even when introduced in the form of liquid into the bowels, the toxic effects are produced much later than with other analogous poisons, as corrosive sublimate. We are, then, led to believe that the acids of arsenic, inoffensive by themselves as phosphoric acid, do not act so much by the arsenic as that they become hurtful in the organism by contracting these toxic forms and new properties, whose nature is undetermined.

§ 144. *Arsenic. Medicine of Economy.*

We know that *local* action on the stomach is nearly always caused by the return of impregnated blood; it is necessary to determine the chemical action of this blood on the organs and parenchyma.

Methods of measuring oxidation.—We judge generally of oxidations by the quantity of their products, that is to say: (1) by urea, a product of decomposition from albuminates; (2) by CO_2, which comes from all anatomical elements whose respiration is active, as muscles; (3) lately Nencki has thought of measuring combustion by the quantity of carbolic acid produced from benzene introduced into the organism.

(a) *Diminution of urea.*—Twenty years ago Sabelin believed he had demonstrated that arsenic is a means of oxidation, and that urea is eliminated in greater quantities than in the normal state. But in 1868, when C. Schmidt

and Sturzwage announced that in the cat there was diminution of urea, and at the same time of CO_2, to verify these contradictory assertions, one of my pupils, Lolliot, instituted a series of experiments which gave me the clearest results in dogs and man, showing relative arrest of combustion. Gubler, who had never made an experiment, reproached me with my change of opinion. Since then, in fifteen years, there has not been a discordant voice. They have agreed even in taking my work and the name *medicine of economy*, which pleased them by its justice and novelty.

Nothnagel alone made an objection. He considered that the animals vomited under the influence of the poison, and were thus reduced to inanition, and consequently a diminished production of urea. But if the animals did not vomit, what becomes of the objection? Lolliot has also been blamed for not giving the exact balance-sheet of nutrition, that is the daily measure of the nitrogen administered to the animals; but the daily quantities of nutriment were rigorously uniform during the experiments, and for from five to eight days previously. De Bök, experimenting on a starved animal, and with receipts and expenditure of nitrogen well equalized, was led to deny all influence of arsenic on the decomposition of albuminates, and consequently the elimination of urea. According to this author, he dealt with *inoffensive* doses—we may even say, useless. In a diametrically opposed sense to the *economizing* action of arsenic, Gaethgens and Kossel endeavoured to establish what Sabelin had indicated twenty years previously, that arseniate of soda produces excessive destruction of albuminates. But at what dose? One centigramme for each kilogramme of body-weight, or sixty centigrammes for man. This was not an experience in therapeutics, it was criminality. The question is, then, decided.

(*b*) *Diminution of carbonic acid.*—The carbonic acid exhaled is also in smaller quantity, as proved by the experiments of Schmidt and Sturzwage on the volatile exhalations. De Meyer and Williams have stated that in the superior animals the lowering of the figures for CO_2 in the blood and in the expired air is constant and considerable. Here is the probable explanation. In consequence of the action of arsenic on the parenchymas, and by lessening of oxidation, the acid products of nutrition which are burned normally remain intact in the poisoning. The blood is thus deprived of a part of its alkalies, and consequently a certain quantity of carbonic acid which is combined with the alkalies as carbonate. Thus the first cause is alteration of tissues; the second the denutritive movement, which is not merely a sequence, it hinders perhaps also the formation of CO_2.

(c) *New method of measuring oxidations by benzol.*—Nencki and Ziegler have discovered an ingenious process for measuring oxidations. Benzol (or benzine) is oxidized in the organism into phenol, pyrocatechin, hydroquinone. Outside the body these operations only take place through atomistic oxygen. The determination of the products of oxidation of benzol in the organism will then be a measure of the quantities of oxygen formed in the tissues. As this quantity is parallel to the intensity of bodily oxidation, to measure the products of oxidation of benzol is thus to appreciate the process of oxidations in the organized body. It is especially the phenol (carbolic acid) whose proportion serves to solve the problem.

Experiments on benzol.—In injecting a rabbit with one gramme of pure benzol, we obtain a proportionate quantity of phenol. If we poison the animal with phosphorus, the phenol disappears. It is the same for copper. For *arsenic*, on the contrary, the phenol eliminated remains at the normal proportion, which seems to contradict all the pre-

ceding data. But Nencki has observed that arsenical gastro-enteritis determines intestinal fermentations, which augment the production of phenol.

Measure of oxidations by lactic acid.—On the other hand, Meyer and Seitelberg (Arch. f. Exper. Path. u. Pharmakol, 1883, vol. xviii.) have remarked that, under the influence of arsenic, lactic acid is formed in the blood, which can but be the result of defective oxidation.

(d) *Temperature.*—As a consequence of the arrest of decomposition of albuminates, people have constantly noted the diminution of general temperature of the body. In presence of this fact, we have no longer to discuss the question of the exciting or hyposthenic action of arsenic. If Trousseau and Graves voted for excitation, it was because, instead of judging with the aid of a thermometer, they founded their opinions solely on the appearances of circulation; that is, on the colour of the face under influence of vaso-motor action.

(e) *Red corpuscles.*—The corpuscles were diminished in arsenical poisoning according to Delpeuch, who demonstrated at the same time that the absorbing power of blood for oxygen was diminished, whilst the leucocytes are scarcely augmented. It is one proof more of the sub-oxidizing action of arsenicated blood.

§ 145. *Depressor of the Circulation and of the Heart.*

Depression of the circulation.—The dominant fact in the action of arsenic is the profound trouble which it produces in the circulation, that is, a considerable depression of arterial circulation. The tension is often so much diminished that circulation is insufficient (Böhm and Unterberger).

Causes of this depression.—The vessels of the abdomen

lose their tonicity, although without the nerves supplying them (splanchnic nerves) being deprived of excitability.

Paralysis of heart.—Besides dilatation of vessels and increased rapidity of pulse, arsenic, at least in frogs, produces paralysis of heart.

Intra-intestinal circulation.—The abdominal circulation is profoundly troubled; the intestine during life is the seat of pain, diarrhœa, and presents profound alteration of mucous membrane, which certainly depends on dilatation of vessels. We may admit that congestion of villi determines transudation of a coagulable liquid, followed by desquamation of epithelium, ulcerations, etc.

§ 146. *Arsenic. Respiratory Medicine.*

In the history of therapeutic uses of arsenic, as in that of the Styrian arsenic-eaters, we have the remarkable phenomenon of its effects on respiration, which becomes less an imperious necessity; need for air seems to diminish. The CO_2 in expired air is lessened, or, in other words, there is relative excess of oxygen.

§ 147. *Local Action on Stomach and Intestine.*

Small doses.—From two to eight milligrammes cause a slight sensation of pain, which the patients often take for that of *hunger*. The gastric juice, saliva, and pancreatic juice are not modified by arsenious acid (Böhm and Schäfer).

Large doses.—When more than one centigramme is taken, after several minutes vomiting of food is caused; later the vomited matters are bilious and bloody, but violent nausea for some hours may precede this. At the same time there is great dryness in the throat, intense smarting and constriction, dysphagia, with a feeling of internal burn-

ing and unquenchable thirst. Abdominal pains begin early and attack all the abdomen, which becomes the seat of meteorism, violent colics, and a diarrhœa often mixed with great quantities of blood; in a word, it produces a series of accidents analogous to cholera.

§ 148. *Arsenic as a Neurotic.*

Fibrillary trembling of muscles is noted in arsenical poisoning; later, paralysis of extremities of motor nerves. The reflex centres of spinal cord lose their irritability, and the paralysis finishes by gaining the respiratory centre.

§ 149. *Arsenicism. Effects due to a Continuation of Arsenical Treatment.*

Digestion is profoundly troubled. Later there are alterations of other organs; the integuments and nervous system are affected. Arsenical œdema commences in the eyelids, but afterwards attacks the lips; the conjunctivæ are reddened. Œdema is generalized; at a later period the skin becomes covered with eruptions, which often ulcerate. The alteration of the blood, general steatosis, and disappearance of glycogenetic function of liver are also to be noted.

Arsenic, then, acts in phthisis by the modification that it impresses on the constitution of the parenchymas, by its function of economy, by its power of relieving dyspnœa, by the depression of the circulation; it has a less secretory power than iodine.

XLIII.

SECOND SERIES. MEANS OF ECONOMY OR MODERATORS OF DENUTRITION.

Several of these medicines constitute true foods. We must distinguish—

(a) *Fats,* comprising the usual neutral fats, cod-liver oil, glycerine, milk; all moderate the consumption of albuminous foods, and favour their assimilation.

(b) *Gelatine,* although nitrogenized, acts in the same way; it enables the organism to profit more from other nitrogenized food.

(c) *Alcohol,* which offers numerous advantages without constituting a regular method of treatment; it is valuable from its action in lessening body changes; it hinders denutrition—witness the diminution of urea in urine and lessened quantity of CO_2 in expired air.

Mode of action on microphytes.—The microphytes do not support a highly oxygenized atmosphere, but at the same time they obtain their supplies of oxygen from the tissues and blood. Fats consume the oxygen of the blood, producing excess of carbonic acid; the supply for the micro-organisms is insufficient. All members of this group act by preserving pre-existing tissues.

§ 150. *Fats in General.*

§ 151. *Cod-liver Oil.*

§ 152A. *Moderating Effects. Indications.*

Except in febrile forms, where pancreatine and bile are not secreted in sufficient quantity, these fatty bodies may be prescribed at all periods, at all times or stages of the malady.

§ 152B. *Glycerine.*

§ 153. *Milk. Milk Treatment.*

In gastritis or dyspepsia of tubercular origin, the milk treatment is badly supported; like glycerine, it is a powerful means of conservation for patients. Casein, although easy of *peptonization*, sometimes becomes difficult of digestion, because of the lactic and butyric acids, which are formed at the expense of lactose.

§ 154. *Gelatine.*

Voit has perfectly demonstrated that this is one of the most evident means of economizing tissue change.

§ 155. *Alcohol.*

1. Alcohol considerably diminishes the quantity of atomistic oxygen disposable for consumption of fats and albuminates; it is a powerful economical agent.

2. By hindering oxygen from using up the albuminates, alcohol finds a triple indication in tuberculosis. If there is fever, alcohol moderates it; if the malady is apyretic, alcohol sustains the strength; in every way it stops body-waste.

XLIV.

THIRD SERIES. NECROPHYTIC OZONISANT SUBSTANCES.

§ 156. *Oxygen.* § 157. *Ozone.*

We may deduce from all the rigorous observations, all the interesting experiments of modern physiology, that we cannot trust to barometric variations to modify respiration, nor to altitudes to diminish oxidation, nor to low countries to augment combustion. We must invoke another cause to

explain the benefit of the climate of altitudes. The inhalation of pure oxygen or condensed air may be dangerous, and the irritating effects of ozone on the mucous membranes are well known.

§ 158. *Necrophytic Aromatic Acids.*

(Benzoic, creosotic, and salicylic acids are all described in the French edition, with physiological actions. The benefits for phthisical patients are very doubtful.)

§ 159. *Creosote.*

Bouchard and Gimbert advise creosote in solution to be taken at meal-times, the dose to be from 40 to 50 centigrammes, and even up to one gramme daily. The preparations recommended are aqueous solutions 1 to 1000, oily solutions 1 to 100, and a strongly alcoholized wine. The benefit is less from antipyretic action, or by modification of nutrition, than by direct effect on the local lesion. Subcutaneous injections of creosote dissolved in oil are not of much utility in phthisis (Bouchard).

§ 160. *Turpentine. The Essential Oil.*

Air saturated with turpentine vapours.—It suppresses the effects of putrid mucus on the respiratory organs; and further, it slows the pulse and respiration. It is an excitant of the central nervous system, whilst at the same time producing depression of respiration, circulation, and temperature. Following Skoda, the anti-parasitic effects of turpentine have been used in the treatment of fœtid bronchitis or pulmonary gangrene. The parasites which cause putrefaction are destroyed by turpentine, which is, as we know, one of the bodies most charged with ozone. There is here a rational appliance to be used against the bacillus tuberculosis.

§ 161. *Sulphur and its Compounds.* H_2S.

M. Nièpce says, "Tuberculosis may be modified or cured by the therapeutic agency of sulphuretted hydrogen."

(The description of sulphur waters, the alkaline sulphides, must be sought in special monographs; the attention of the reader is, however, specially directed to the hot springs and sulphur lakes at Rotomahana, New Zealand.) The physiological effects are those of H_2S. The treatment comprises, besides breathing gas in the bath, or by inhalation, an internal use of the thermal waters. All these effects united, without counting on altitude, may serve as a basis for the treatment of phthisis with predominance of bronchial catarrh, on condition that it is apyretic, or at least at a period uniform with regard to thermic oscillations. Like all cold or thermal mineral waters, they favour hæmorrhagic congestion, although producing generally a notable quieting of circulation; sometimes also return of physical strength, and an amelioration in what is called, vaguely, general condition. Locally we see the secretion diminished or modified up to the point of losing its purulent character; but the cavernulous or cavernous *râles* persist, as well as signs of peritubercular engorgement recognizable by dulness. The catarrh does not disappear; it ceases to be infecting. It is, shortly, the secretory element of the bronchi, that is, the catarrh, which is amenable to sulphurous waters; but we must not forget it is the result of peribronchial bacillary lesion. We have not to establish a parallel with primitive catarrh, which follows a happy course under the influence of sulphuro-thermal treatment. The catarrh which accompanies or follows asthma receives but little more benefit than tubercular bronchitis equally on account of the cause not being removed.

§ 162. *Sulphur. Sulphurous Acids. Hypo-sulphites.*

XLV.

FOURTH SERIES. MINERAL FOODS.

Voit (*Pflüger's Archiv*, 1883) describes *mineral abstinence*. Inanition does not produce a correlative change of elements in the organism, whilst by mineral abstinence the saline contents of the organs diminish. Thus, by alimentation without albumen or fat, the brain is not impoverished in albumen or fat, but if the saline matters are wanting it loses salts. It is not indifferent whether the nervous substance does or does not possess its physiological quantity of mineral salts; in the latter case we must count on pathological manifestations. What Voit says as to the nervous substance applies to all organs. That mineral abstinence is dangerous, and reciprocally that inorganic compounds may have their utility, is the starting-point of the conception of mineral medications.

§ 163. *Phosphorus and Lime.*

The utility of lacto-phosphate of lime seems to be clearly demonstrated.

§ 164. *Phosphorus and Hypo-phosphites.*

§ 165. *Chloride of Sodium.*

Physiological effects and over-oxidizing action. — It causes a more marked elimination of urea, but is apt to produce hæmoptysis if used medicinally.

§ 166. *Alkalies and Alkaline Waters.*

XLVI.

APPRECIATION OF METHODS OF TREATMENT. NATURAL COURSE OF THE MALADY. PROGNOSTIC VALUE OF BACILLI.

BEFORE attempting to appreciate methods of treatment, and the application of pharmaco-dynamic measures, there are two important questions to be solved: one is relative to the natural course of the malady, its progression and retrogression—without this notion one might be tempted to attribute to the remedy what really belongs to the disease; the other relates to the prognostic value of the revealing signs, or rather *the* vital sign—I mean numerical oscillations that the bacilli may present in their rapid or long evolution. Is there in it, that is to say the bacillary infection, any criterion for us to fix the stage, or degree, the cure, or dangerous relapse of phthisis? If it is thus, we have then to consider the medications and formulate the therapeutics of phthisis.

§ 167A. *Prognosis according to the Course of the Malady.*

The course of phthisis has nothing regular about it, and no prevision can be established from its mode of development. With the exception of granular phthisis, which nothing can arrest in its fatal evolution, we can say that ordinary phthisis may take the most diametrically opposed courses. Thus, after having started brusquely with bacillary broncho-pneumonia, it stops rapidly in its progress; the softening and ulceration of the tubercular mass are effected, and the cavity may persist for an indefinite time. Unhappily this is not the habitual case. In general, phthisis, at the first onset massive, becomes rapidly invading and

mortal. Here is an inverse and more frequent eventuality: the malady exists for several months or years under the form of a considerable excavation; all at once, without known reason, a new bacillary invasion, starting from this cavity, appears in the neighbouring parenchyma, which caseifies and rapidly ulcerates; it is chronic phthisis, terminating by a subacute, galloping consumption. A third case may occur—it is the only favourable one. This is chronic phthisis, remaining so during long years, and finishing by taking the fibrous form, which, without being curable, is indefinitely compatible with life. We see the phthisical, who for twenty or thirty years have been attacked with tuberculosis, die from old age, or at least in the period of old age. Of these three eventualities, nothing can indicate which is about to happen. It is not until the cavity has become sclerous, when the thorax has contracted, fever been wanting for long years, and the loss of flesh completely arrested—it is not until then that one can establish what I shall call the prognostic diagnosis.

§ 167B. *Prognosis according to the Origin of the Malady.*

Three kinds of indices of curability have been described: (1) the forms and different modes of origin of phthisis; (2) functional and physical signs; (3) a third indication seems alone to be able to reveal all the truth; this is the multiplication or disappearance of the virulent agent. It is the bacillus which, *à priori*, should decide the lot of the patient.

I. *Origins of phthisis.*—Phthisis has, they say, pathological causes or preparatory conditions of physiological order; in reality there exist phthisogenic maladies, and there are individual causalities.

Phthisis of pathological origin or from diathesis.—At first sight one cannot comprehend how a malady or general

constitutional state or diathesis can determine phthisis, which definitely is a virulent malady. The organism must be attacked, in a manner, to prepare it for the bacillary invasion. This, then, is the influence people have attributed to the scrofulous diathesis, to far advanced gouty disease, but especially to diabetes. According to these ideas, there is *scrofulous phthisis*, which will be slow in its evolution; *arthritic phthisis*, which may pass into an indifferent condition; and *diabetic phthisis*, which will infallibly lead to death.

But there is one objection to make to these subtle distinctions. Scrofula, which is exterior tuberculosis, or phthisis itself with another localization, if it has proceeded tacitly for some time as scrofula, a time comes when it takes the character of an acute tubercular affection; it is the end of two maladies which are really but one. There is, then, no prognosis to be formed for this variety of phthisis.

Let us see how the arthritic patient, who has become tubercular, behaves. Jaccoud says, with reason, that arthritic phthisis is phthisis in a gouty patient. If the invalid does not complain of his gout, it is absolutely impossible to distinguish his illness from common phthisis. People have supposed it has a slower course, more rarely marked by hæmoptyses; but all these signs are illusory. There is one terrible formula—all the world is equal before the bacillus.

The same reflection applies to diabetic phthisis. It may be insidiously developed, that is true. I have seen numerous examples of it; and that is why I have recommended in my Academic Memoir of December 4, 1883, the examination of the *crachats* from diabetic sufferers attacked with bronchial catarrh; but it is not rare to see the scene suddenly terminated by mortal hæmoptyses. Thus the diverse forms of origin for phthisis do not exist; they are not there to be recognized, or for the issue to be foreseen from them.

Hereditary or acquired phthisis.—The same remarks apply to hereditary or acquired phthisis. *A priori* it seems that one must be fatal, the other alone curable. It is not so. I have seen in several phthisical families the malady definitely arrested in one or more members. I shall quote two striking examples. One, a lady, who is now seventy-three years of age, born of a phthisical father, and has shown, for the twenty years I have attended her, all the signs of a pulmonary cavity. She has had two daughters, of which one has succumbed at thirty years of age, after having lost two children from meningitis; the other daughter, aged thirty-eight years, has been struggling for twenty years against ulcerous phthisis. The poor grandmother has seen one child and two grandchildren perish, and she lives always, as people say, with one lung. The other case relates to three daughters born of tubercular father and mother. Of the three children, one has perished from laryngeal phthisis; the second has escaped with incurable chlorosis; the third has for a long time shown signs of a cavity, which does not, however, make any hurtful progress. Thus hereditary phthisis may be fixed, localized, limited, exhausted, so to speak, on the spot, like acquired phthisis which has been contracted by direct contagion.

II. *Prognostic symptoms and signs.*—What ought one to conclude from local phenomena? What can we infer from functional troubles of the whole organism?

Hæmoptysis, the first and often also the last sign of tuberculosis, has but a limited influence in the issue of the malady. It produces more alarm than disasters. If hæmorrhage does not lead to immediate danger, it may be cured without interfering with the recovery of the patient, who is cured often, very often, after considerable and repeated losses, which, however, are during the first period of the malady.

It is not the same at the termination; hæmoptysis at the ulcero-cavernous period is due habitually to the rupture of intra-cavitary aneurisms; it is a mortal incident or index. Ulcerations of the larynx have a dangerous signification; they may become mortal if they attack the epiglottis; the patient dies from inanition.

Fever, emaciation, chloro-anæmia, urinary secretion, charged with excess of products from denutrition, constitute together, or even separately, a bad state of affairs. Fever, however, need not be absolutely feared; it may be cured or may stop spontaneously, but leaves after it an exhaustion which is added to the other signs of denutrition. The patient who becomes emaciated is in a bad way; but if after this loss he recovers his strength and flesh, be sure the disease has not made further progress. It is the same if this result is obtained by medicines of economy or fattening diet. In general, all treatment which stops what is vulgarly called ethisis (filtration) should be regarded as good; the arrest of denutrition is its distinctive mark. Chloro-anæmia, which, far from being limited to hypoglobulism, strikes the organism *totius substantiæ,* inspires me generally with grave inquietude; much more when, as is frequently the case, it is accompanied with profound perturbation of digestion and assimilation of food. Beware of this state of decolourization of blood and tissues; always examine the urine from the triple point of view of composition as to urea, phosphates, and especially the quantity of phenol resulting from the oxidation of benzol. This is an exact measure of denutrition. Nencki has indicated it, and I have been able to verify his statement in all phthisical patients who are losing flesh. Thus the physico-chemical signs of organic change should enter into our consideration before everything; they are of more value than any other local sign, or than pulmonary hæmorrhage; more important

than the form or origin of the malady. There remains yet but to measure the progress or retrogression of the malady by the bacillus, that is, by the infecting agent itself.

§ 168. *Prognostic Value of the Number and Form of Bacilli.*

Having once recognized the semeiotic value of bacillary *crachats*, their absolute signification from a diagnostic point of view, and their ætiological importance, people have not hesitated in ascribing to the bacilli, by their variations in form and number, the power of regulating the prognosis of the malady itself; its eventualities of such different degrees of importance being governed solely by the state of the parasitic population.

(a) *Diverse forms of bacilli.*—Immediately after the discovery of the bacillus, Balmer and Fräntzel sought to demonstrate that, when one finds the bacilli small, curved, and furnishing but a small quantity of spores, the malady progresses slowly, or stops entirely; on the contrary, voluminous bacilli, laden with spores, indicate grave cases; but Fräntzel has never been able to verify these statements. When the bacilli are numerous in the *crachats*, they present all possible forms in the same preparation; they are large or small, with or without spores, and this has no relation to the gravity of the malady. Fräntzel has seen a phthisical patient, cured, whose expectoration, at first free from parasites, afterwards contained very large bacilli charged with spores. In three cases which became mortal, only very short, though voluminous, bacilli were found. In a word, the dimensions of the bacilli do not allow us to establish a prognostic conclusion.

(b) *Number of bacilli.*—The number of bacilli should have more marked importance from a prognostic point of view; the prognosis is aggravated by a large number, so

that in florid phthisis the colonies are immense; that is the opinion of Balmer, Fräntzel, and Müller, etc. To appreciate thoroughly the quantity of bacilli, Fräntzel has established classes. No. 1 answers to very few bacilli, which are wanting from time to time; No. 2 to a less collection than that of other tissues; No. 3 indicates very marked predominance of bacilli. Gasky improved further on these minutiæ. He divided the bacillary collections into ten series, according as the entire preparation contained but one to four bacilli, or four to six, or seven to twelve, etc. The more or less advanced terms of the series should indicate the intensity of the danger. This we may assert with Fräntzel, Lichtheim, Ziehl; but it appears clearly from my personal researches that the multiplicity of the parasites does not in any way indicate the gravity of the lesion, nor allow us to foresee such accidents as hæmoptysis or pneumothorax. If the richness of the tissues in bacilli is in a certain relation with the intensity of the disease, it is not the same for bacilliferous *crachats*, which do not in any way allow us to judge of the parasitic contents of the organs. Thus the internal surface of the cavities may, perhaps, be covered with bacilli, though such cavernous walls, when they are hard, scarcely contain them. Inversely, when the substance of these surfaces results from recent fusion of caseous masses, numerous bacilli appear in the *crachats*. It seems from this that expectorated matters much charged with bacilli are the sign of a galloping consumption; however, there exists no fixed rule in this respect. But if there is no conclusion to be drawn from a bacillary invasion more or less complete, on the other hand we have the right to consider the permanent disappearance of the bacilli from the *crachats* as a sign of cure, although the other physical signs continue to persist. We must add a last consideration. It seems that a larger quantity of bacilliferous *crachats* should

make us fear exceptional virulence and gravity. We may say here, again, that such a prognosis based upon this fact would often be incorrect.

(c) *Résumé of observations.*—In twelve cases observed by Gasky, the results showed that all conclusion from the number of bacilli was impossible. To offer an opinion, formed from this basis, as to the ulterior course of the malady would be a vain presumption.

XLVII.

TREATMENT OF MASKED OR BRONCHO-PNEUMONIC FORMS OF PHTHISIS.

§ 169. *Revulsives.*

PAINS and dyspnœa are, without doubt, relieved and lessened by revulsives; applications of tincture of iodine, or a flying blister, to the affected part, may often cure fixed or erratic pains which occupy different points of the thoracic cavity. Their benefit in phlegmasies of bacillary origin has not been demonstrated.

§ 170. *Of Surgical Derivatives and Metastasis.*

The evidence seems to be that the doctrine of metastasis is altogether mythical, and that we ought to treat scrofulous lesions or anal fistulæ with a view to immediate cure, without regard to ulterior consequences.

XLVIII.

TREATMENT OF INITIAL FORMS.

§ 171. *Tubercular Chlorosis.*

HYDROPATHIC treatment and iron, so frequently prescribed, have done much injury in this malady. The exclusive

treatment by *raw meat* is the best to commence with, as said Professor Lasègue; later with the gradual addition of a single albuminoid food. Good Bordeaux wine or English beer may be also added.

§ 172. *Treatment of Tubercular Dyspepsia.*

Régime in general.—Without fat or farinaceous foods, denutrition is inevitable, because, to supply the deficit of carbon eliminated, the organism is obliged to use up its albuminates to take the place of the fat which is wanting in the diet. The proteid matters are decomposed to furnish fatty food, and are thus in part exhausted; the assimilation of remaining nitrogenous materials is singularly reduced; they are burned, and more easily destroyed than in presence of fats and farinaceous substances; excess of urea in the urine may be regarded as the proof of this denutrition. Fats and starchy foods must be prescribed to phthisical patients in sufficient quantity to repair the waste of carbon; from 60 to 100 grammes of fat, and 500 to 600 grammes of feculents, as bread, pastry, or dry decorticated vegetables, etc. To obviate loss of nitrogen, 120 grammes of azotised food will be sufficient; it must consist of meat, game, or fish, and be made as palatable as possible. To preserve the appetite, it is really indispensable to vary the forms of diet indefinitely, but to rigorously maintain the quantity of hydrocarbons in excess of that of the nitrogenous food. It must be noted that we have in view the patient who is relatively in good health, and whose digestive functions are regular.

Actual treatment of dyspepsia.—This dyspepsia, ordinarily neuro-asthenic, is accompanied by persistent flatulence, dilatation of the stomach, gaseous distension of intestines, and, as a consequence, mechanical dyspnœa following the

previously troubled respiration. I prescribe (1) Vichy water, a tumbler half an hour before each meal, to favour the secretion of gastric juice.

(2) At the beginning of the meal an absorbing powder:

Precipitated chalk	1 gramme
Calcined magnesia	2 grammes
Powdered rhubarb, or „ calumba	10 to 20 centigrammes.

(3) If there is constipation, the patients take a teaspoonful of linseed (not the powder) each meal-time.

(4) The diet is neither to be uniform nor systematic: it will be composed of meat, game, fish, with dry decorticated vegetables, salads, preserved or fresh fruits.

(5) To favour the passage of this alimentary mass into the intestine, and to replace gastric digestion by intestinal digestion, nothing is more useful than warm stimulant drinks, like tea or warm alcoholized drinks, which are so much superior to wine, which easily turns sour, or to beer, which ferments in the stomach. Aërated waters and iced drinks are to be avoided, as often hindering peptonization.

(6) Lastly, when the dyspepsia resists the use of these diverse dietetic and therapeutic means, the best treatment consists in washing out the stomach with a warm aromatic infusion, or with warmed Vichy water. As the end of a certain number of these applications, the stomach, freed from the mucosities which cover its surface and provoke putrid fermentation of food, finds itself, so to speak, uncovered; and once more in contact with food, which, particularly when stimulating, provokes the secretion of gastric juice, the stomach again begins to discharge its functions. In future we may permit all the food that the patient asks for. The same rules apply to the treatment of anorexias with or without fever.

§ 173. *Treatment of Cough in Phthisis.*

(Professor Germain Sée recommends morphia as the remedy *par excellence*, and gives a careful account of its advantages and inconveniences. In hæmoptysis, morphia constitutes the most powerful means of diminishing the excitability of the mucous membrane which provokes the cough, and for setting the bronchi at rest. Chloral and paraldehyde are not to be recommended; they have not the favourable effects of morphia on respiration and the bronchial secretions.)

§ 174. *Treatment of Paroxysmal Cough with Vomiting.*

Aconitine.—Duquesnel's aconitine, in doses of one quarter of a milligramme each day, often suffices to calm previously obstinate cough, and to put an end to the vomiting which follows it.

Tincture of iodine. Alkaline iodides.—Iodine in very small doses has been long recommended in the treatment of vomiting. In prescribing five to ten drops of tincture, with the addition of ten centigrammes of iodide daily, I have often observed a double effect—cessation of vomiting, and especially the transformation of the dry cough into one more easy, more mucous. It is perhaps by the last action that we stop the vomiting which results from the cough. Washing out the stomach has been tried by Debove, Dujardin-Beaumetz, and myself, with some success.

§ 175. *Treatment of Hæmoptysis.*

Hæmoptysis of initial phthisis is curable, even if the loss of blood is very considerable. If we have to deal with ulcerous phthisis, which is at all times of its evolution known by the hæmorrhagic form, we may still conceive the hope

of stopping these incessant losses. But when hæmoptysis comes tardily in cavernous phthisis, it is very rare not to depend on the rupture of aneurismal vessels developed in the walls of the cavity, or already opened into the tubercular cavity itself; in these conditions the most energetic hæmostatic means are ordinarily impotent. The treatment of curable hæmoptysis comprises entirely distinct methods; here is the enumeration and analysis.

1. *Vomitive medication,* which is only mentioned to be condemned, although practised by illustrious men, as Trousseau and Peter. Ipecacuanha and tartar-emetic are the agents they selected.

2. *Turpentines.*—Before all other means, I prescribe turpentine in the form of syrup, or pearls, containing half a gramme of the essence in each. It is important to add a small quantity of opium to the turpentine.

3. *Ergot of rye. Ergotine.* — Thirty-eight years ago I gave a detailed account of the hæmostatic powers of ergot in an inaugural thesis. Ergotine in hypodermic injections is one of the best remedial measures against hæmorrhage; it should be followed by the hypodermic injection of a centigramme of morphia, which has the desirable effect of modifying the respiratory rhythm and diminishing the extent and number of respirations, and especially by suppressing the paroxysms of cough which provoke the bloody expectoration. Opium or morphia should be associated with all other medications.

4. *Tannic* or *gallic* acid is of doubtful utility.

5. *Sulphuric acid* presents several advantages after turpentine or ergot.

6. *Perchloride of iron.*—Clinically iron must be considered as producing uterine and pulmonary hæmorrhagic congestion, although physiologically its hæmostatic power is considered proven by the fact of its slowing the action

of the heart and diminishing the amplitude of arterial pulsations.

7. *Alcohol in large doses.*—This powerful hæmostatic means, so frequently used in obstetric practice, has been successfully employed for the treatment of hæmoptysis even of very long standing.

Ice or warm drinks.—Both these methods are of very doubtful utility.

§ 176. *Treatment of Catarrhal or Pseudo-asthmatical Forms.*

The best method of treatment is iodide of potassium, in doses of two grammes daily, with the addition of five centigrammes of extract of opium.

Tannic or gallic acids are both useful in bronchorrhœa by diminishing secretions of mucous membrane.

Antimonials, although used as expectorants, are much inferior to apomorphia.

Apomorphia, like pilocarpine, singularly increases bronchial secretion, so much so that mucous *râles* are to be heard all over the chest; but it is remarked that during this time the vessels remain in a normal condition, and that, contrary to the ancient opinion, hyper-secretion takes place without congestion.

§ 177. *Treatment of Sweating.*

(In 1870, Sydney Ringer, and in 1873 Fräntzel (and at the same time Vulpian), recommended the employment of atropine in the treatment of phthisical sweating. In the form of pills, according to the formula of Vulpian, atropine may render real service without presenting the least danger. Subcutaneous injections of atropine, described by several authors, cannot be employed without grave inconvenience. The classical work of Ringer may be

consulted for the physiological action of atropine, and the antagonism between it and the two alkaloids, muscarine and pilocarpine, is clearly shown by the researches of Vulpian. He administers atropine in pills, containing one milligramme in each. The results have been prompt and efficacious.)

§ 178. *Treatment of Febrile Forms.*

(The precise experiments of Hallopeau with regard to kairine are described, also the therapeutics of quinine and salicylic acid; but M. Sée gives as his conclusion that it is best to use sulphate of quinine, in doses of one gramme, each morning, fasting. This is taken for three days, then an interval of three days, and a second and a third similar attempt, in series of three days, is to be made. If quinine does not succeed, arsenic should be prescribed in the indicated forms.)

XLIX.

TREATMENT OF FINAL PERIODS.

§ 179. *Treatment of Laryngeal Phthisis.*

The ordinary precautions against laryngeal catarrh, such as avoiding currents of air, dust, irritant vapours, fumes of tobacco, etc., must be taken. Afterwards, in early stages, we prescribe: (1) Inhalations with steam spray producer, of tar water, or sulphurous waters, as Cauterets. The arsenical waters of Mont-Doré and La Bourboule are much employed for the same purpose. (2) Direct applications to the inflamed parts, with little sponges dipped in iodized glycerine or laudanum (Fauvel). In the ulcerous or œdematous period, when we see manifested very painful

dysphagia, severe pain in the region of arytenoid cartilages and ears, also very rebellious salivation, we must have recourse to the hypodermic injection of morphia, which is the surest method of arresting salivary secretion, rendering digestion easy and facilitating respiration.

Inhalation of sedative liquids, like solution of potassium bromide, with cherry laurel water and morphia, act with efficiency against the painful symptoms, but much less than subcutaneous injections of morphia.

Antiseptic inhalations, as perchloride of mercury in solution 1 to 3000, have given good results (Fauvel). But Schnitzler has shown that we should prefer iodoform fumigations, which ease pain and are really antiseptic.

We must mention boracic acid, indicated recently by Schech (*Deutsche Woch*, June 12, 1884). Scarifications of œdematous parts constitute a dangerous operation. Without giving the least relief, they singularly favour the development of traumatic ulcerations, which, added to the pre-existing tubercular ulceration, end by attacking or destroying the perichondrium, or bones of the larynx. We must, with similar severity, forbid cauterization with nitrate of silver or perchloride of iron as spray.

§ 180. *Treatment of Diarrhœa and Tubercular Ulceration.*

We have first to attack nervo-muscular excitability; secondly, to remedy the loss of digestive intestinal liquids, and the products of digestion; thirdly, to modify the morbid action of intestinal glands, which are often rendered bloodless by amyloid degeneration of arteries, or they may be ulcerated by tuberculosis.

Nervo-motor medicines.—Opium, morphia, belladonna of which the actions are well known and frequently used.

Reparative diet. Raw meat. Alcohol.—The most certain mode of stopping diarrhœa, when opiates do not succeed, is

the use of raw meat, which must be prescribed in an exclusive manner, the daily dose being three hundred grammes, suspended in warm broth. At the same time brandy or rum, diluted with slightly sweetened water, is ordered as an habitual drink; suppressing the use of wine, beer, or mineral waters.

Raw meat is readily peptonized in the stomach.

Modifiers of secretion. Astringent medicines.—Subnitrate of bismuth or prepared chalk, often added to preparations of opium, is most frequently eliminated by increased peristaltic action without producing the least effect.

Nitrate of silver should be avoided, as also tannic and gallic acids, which are of little utility. The turpentines combined with opium appear to me of much greater service.

Inert deobstruants.—When diarrhœa alternates with constipation, I often prescribe such inert bodies as grains of linseed, or plantago psyllium, and at the same time a mixture containing small doses of opium and belladonna.

In *incoercible* diarrhœa a combination of quinine and opium has appeared to me to sometimes present certain temporary advantages.

TRANSLATOR'S NOTES.

THE seventh part in the later sections is little more than a summary of the views expressed by Professor Germain Sée in his work.

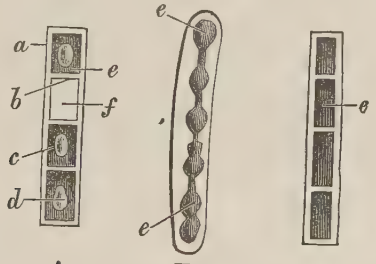
The English reader will find that the classical books on therapeutics in our own language contain almost all the information given in the French edition.

The practical steps to be taken with regard to the prevention and treatment of phthisis are obviously still capable of improvement.

POSTSCRIPT.

THIS translation was completed by the end of August, 1884, but was not published on account of circumstances over which the translator had no control, he being absent from England. Since then the subject has been constantly before the profession, but no really material advance has been made. The fact of the centigrade system of weights and measures being retained will scarcely require an apology.

Professor Ray Lankester (*Nature*, December 25, 1884; Lecture on Bacteria the Germs of Putrefaction and Disease, University College, January 14, 1885) describes the Bacillus of Tubercle as never giving rise to spores by endogenous formation, its multiplication is by fission or breaking up into segments capable of growth. The protoplasm constituting the bacillus is always arranged in moniliform masses. The figure is that given by Mr. Lankester in illustrating his statements.



A. Diagram of bacillus subtilis of hay infusion during sporulation.
a, sheath of bacillus.
b, transverse septum.
c, coat of a spore.
d, contents of a spore.
e, protoplasm surrounding spore, which disappears entirely when the latter is fully formed.
f, empty or sterile segment.

B. Tubercle bacillus; the protoplasm is arranged in moniliform masses,
e, which were erroneously called "spores" by Koch.

C. Diagram of hay bacillus in a vegetative state; the protoplasm is arranged in block-like masses,
e, comparable to the moniliform masses of B.

According to Lankester, at present only three bacilli are proved to produce *resting spores* in the manner described in text; of these the best known are *Bacillus subtilis* and *Bacillus anthracis.*

In his letter to the editor of *Nature,* Mr. Lankester points out the difficulties and uncertainties connected with some aspects of the question, and rightly insists on a knowledge of the methods and opinions of the botanists. We may note that almost all his authorities are those referred to by M. Sée. It is to be regretted that the tone of the letter is hostile to Professor Koch. "Our knowledge of the bacteria is in its infancy," and any mistaken observations of that gentleman must be corrected by subsequent observers.

In the chemical world considerable attention has also been given to the subject of the bacteria and bacilli. Ordinary methods of water analysis may receive some assistance from this point of view. Mr. Cassal and Dr. Whitelegge have recently issued an interesting little *brochure* on the usual modes of examining potable waters.

The article "Ferments and Fermentations," by M. A. Henninger, in the *Agenda du Chimiste* for 1883, may be recommended as a valuable contribution to the knowledge of this subject.

Judging from the recent debate at the Royal Medical and Chirurgical Society, English authorities do not give much support to the doctrine which attributes certain contagious diseases to *special ferments* which have been secreted by cells modified through impaired nutrition or otherwise. This specialization of function for the cells or masses of protoplasm has been compared to that of the glandular elements which form Pepsin, Ptyalin, or Pancreatin.

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